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SYMPOSIA ON

I. Tumors of the Oral Regions

HAMILTON B. G. ROBINSON, D.D.S., M.S.
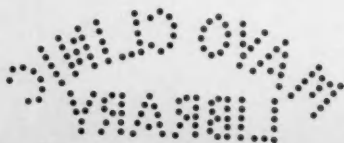CONSULTING EDITOR

II. Modern Practice in Endodontics

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SYMPOSIUM ON MODERN PRACTICES IN ENDODONTICS

Foreword

Dentistry is in one of its rapid periods of development. From humble beginnings with the barber-surgeons, through establishment as a profession in the mid-nineteenth century, dentistry has evolved to a position of responsibility in the health service professions. The dentist of the 1850's would have difficulty in comprehending the field of the dentist of the 1950's, and indeed many laymen do not yet understand the scope of modern dentistry. Added to relief of dental pain and replacement of teeth has been a broader responsibility for the health of the entire oral region, as well as the prevention of dental and oral diseases.

The tissues of the oral region are susceptible to diseases caused by living organisms, chiefly bacteria, viruses and yeasts, by variations in genetic makeup, by disturbed metabolism, by injury from physical, chemical or radiant energy, by variations in the growth process, and by some yet unmasked causes. Within this last group of diseases are the tumors, some benign and others, the killers, malignant. The dentist has a responsibility in this area of health and disease which he dare not neglect. He must be aware of cancer, know how to diagnose it and be familiar enough with the methods of treatment and the outcome of treatment to advise his patients. He must be capable of aiding the cancer therapist and of assuming the major role in rehabilitation by prosthetic aid.

There are many dentists whose background is such that they have neither recognized their obligation nor been trained to accept responsibility for diagnosis of neoplasms. Fortunately, each year more and more dentists are learning to aid in the battle against oral cancer. Some three thousand new dental graduates arrive on the scene annually with an educational background in the field of cancer which they acquired in dental schools. The National Cancer Institute recognized the importance of the dentist in the cancer war and began, almost ten years ago, to give each qualified dental school $5000 annually to aid in their cancer teaching programs. All dental schools have taken advantage of this aid. In addition, more and more dentists

who graduated earlier are learning to accept responsible places on the cancer fighters' team through reading, refresher courses and lectures.

This symposium is designed to give a general picture of the oral cancer problem. It reviews the oral neoplasms and their tumor-like counterparts region by region. It gives some views on etiology. It discusses treatment by various methods and the dental role in post-therapy prosthesis. There are some points upon which the participants disagree slightly with each other, although all are thoroughly qualified and direct their discussions toward the ultimate goal of preserving human health and life. It is interesting that of those discussing the diagnosis, etiology and progress of oral tumors, all are dentists, all are Fellows of the American Academy of Oral Pathology and all are Diplomates of the American Board of Oral Pathology. Dentistry has within its broader field a core of individuals with special interest in disease processes and their diagnosis. Their knowledge in the field of cancer is not inconsiderable.

In addition to these persons whose abilities lie in the field of pathology, Dr. Arthur James, a surgeon and Diplomate of the American Board of Surgery, has explained cancer therapy. His clear discussion not only evaluates surgical approaches but also the various types of radiation, including gold and cobalt implants in which he has pioneered. Dr. William Heintz presents the intraoral prostheses which are so valuable in allowing the cancer patient to function more normally and to enjoy living after therapy. Mr. Joseph Bitonte, a university graduate in dental technology as well as in engineering, teaches the technique of extraoral prosthetic restoration.

While no single text or symposium on oral cancer is complete, the editor believes that his contributors have prepared an unusually comprehensive and understandable symposium. It should serve to refresh the minds of those who have been accepting their responsibility in the field of oral cancer and to stimulate those who have not yet participated fully so that they may join the army of cancer fighters who combat this disease—a disease which will take more than 242,000 American lives this year, and every year until the tide of battle changes.

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Neoplasms and "Precancerous" Lesions

of

the Oral Regions

HAMILTON B. G. ROBINSON, D.D.S., M.S.[*]

The dentist is confronted with a variety of benign and malignant neoplasms, with "precancerous" lesions and with lesions which must be differentiated from the them. Although the malignant tumors are much rarer than benign tumors and other oral lesions, they cannot be considered rare, or in any sense unimportant. Each year 6000 to 7000 Americans die from neoplasms of the oral regions, and at least three times that many malignancies must be estimated to be present and diagnosable in the mouths of dental patients or potential dental patients in this country. What do we know about these tumors?

The cause of cancer is still unknown although science is directing much of its resources toward discovery of the cause, prevention and cure of malignancy.[6] Usually the discovery of the cause of a disease points the way to prevention and effective treatment, but science does not wait to develop therapy in this manner. Perhaps the most striking example of effective control of disease preceding discovery of cause is in the prophylaxis of smallpox by vaccination long before the errant virus was recognized. So with cancer—surgery is being improved, aided by antibiotics in a way that anesthetics aided in the previous century; radiation therapy is increasing its effectiveness with greater emphasis on radioactive isotopes that may be more definitely controlled and on higher levels of intensity that may be more effective; and chemotherapy is gaining recognition following the encouraging results in treatment of leukemias.

THE ETIOLOGY OF CANCER

There are many facts that have been gained in the search for the cause of cancer. What are some of the known facts and some of the theories?

One of the oldest theories of cancer causation blamed irritation.

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Later the investigators became more specific and cited heat and combustion products, then coal tars and finally specific hydrocarbons. Some of these hydrocarbons are so carcinogenic that they can be used to produce neoplasms, almost at will, in susceptible animals. The current discussion of smoking and cancer revolves around the carcinogenic effects of the combustion products of smoking. From statistical studies of the smoking habits of lung and oral cancer victims, and from experimental studies of the effects of tobacco smoke on animals, there appears to be little doubt that smoking increases the probability of dying from cancer as compared with other causes of death. The likelihood that smoking increases the possibility of oral carcinoma cannot be discounted. The present stage of discussion suggests that each individual must decide whether smoking is worth the definite but undetermined risk.

Embryonal rests left behind during development were proposed by Cohnheim in 1877 as possible sources of neoplasms. The frequency of epithelial rests in the periodontium raises the potential risk of neoplasia in the oral regions, although these nests of cells doubtlessly are more important as sources of odontogenic cysts than as causes of tumors. The ameloblastoma may arise from such sources, the odontogenic origin of these tumors appearing to be well established.[4]

Heredity is a factor which may contribute to cancer susceptibility. Despite the development of cancer in genetically susceptible strains of animals, environmental factors appear to overshadow genetic factors as causes of tumors in man. Hormones appear to influence certain types of neoplastic diseases very strongly. In particular, breast, prostate, gonad, and endocrine gland tumors are definitely influenced by hormones and someday may be controlled by endocrine therapy.

Viruses have been shown to cause neoplasms in fowls and in some mammals but clear proof of virus etiology of human neoplasms never has been established. There are a number of investigators who believe that viruses do cause malignancy in man but those viruses have not yet been isolated. The burden of proof still is on those who claim this etiology for human cancer.

The field in which cancer develops probably is as important as, or more important than, the triggering mechanism. It is possible that some change occurs in once normal cells that makes them susceptible to a carcinogen. This may be some intrinsic factor, deep within the cell and locked up with the very secret of life, or it may be one of the influences which also helps control general body growth, hormonal or nutritional for example. Such altered cells may then be susceptible to hydrocarbons, actinic rays, physical trauma, or any other extrinsic factor. Kreshover's studies[3] of the reaction of the oral mucosa to hydrocarbons during stages of nutritional deficiency support such a

view, as does the clinical observation that the occurrence of oral cancer increases in women with Plummer-Vinson syndrome. On the other hand, continued irritation from coal tar products, or actinic rays, sufficient to produce leukoplakia or solar cheilosis, may so alter cells that they may become "tumor cells" if further influenced by either extrinsic or intrinsic factors.

"PRECANCEROUS" LESIONS

The term "precancerous" holds different meanings for different people. To the bench pathologist it may mean only that the lesion shows dyskeratotic changes in microscopic section; to the clinician it may indicate that the lesion falls within a group of changes loosely associated with possible malignant growth; to the patient it may signify the doom of death. One cannot hope to change all this by discussion, but an unemotional review of this situation is in order. In part, because white plaques appear along the borders of certain oral cancers, particularly the type illustrated by Gorlin on the buccal mucosa (see p. 666), it has been concluded that leukoplakia is a "precancerous" lesion. Whether the white lesion preceded the cancer or not is seldom established. It is true that on occasion leukoplakia is observed without clinical or microscopically demonstrable malignant change and that carcinoma develops later in the identical lesion, but how much more frequent is that occurrence than the development of carcinoma directly in a normal-appearing region of oral mucosa? In raising this question there is no intention to belittle the fact that leukoplakia is a warning sign indicating activity of an irritating factor that may be carcinogenic.

Oral pathologists who place their emphasis on the morphologic picture, as seen in a microscopic section, have attempted to separate white, keratotic lesions of the mouth into those showing dyskeratosis and those showing only increased keratosis.[1] The former are considered true leukoplakia, the latter hyperkeratotic lesions or "pachyderma oris," a term of questionable ancestry for the oral mucosa is not dermis and to speak of "abnormally thick skin" of the mouth is incongruous. Moreover, to make this differentiation requires biopsy and histologic examination, a procedure which many competent clinicians consider unnecessary if the history, clinical appearance and course are favorable and if the apparent irritation can be eliminated and regression is observed. In how many white lesions of the mouth, clinically diagnosed as leukoplakia, as differentiated from lichen planus, cheek chewing, drug burns, and white sponge nevus, has carcinoma developed? Are we being over-zealous in our diagnoses and our use of the term "precancerous"?

We must consider the potentiality of leukoplakia, whether we are using the term clinically as applied to keratotic white lesions or histologically as applied to dyskeratotic lesions. In either case irritants are active and the same irritation which causes epithelial cells to mature more rapidly (hyperkeratosis) or abnormally (dyskeratosis) may activate carcinoma if the tissue is susceptible to malignant change. For this reason, it appears that the most important procedure, wher-
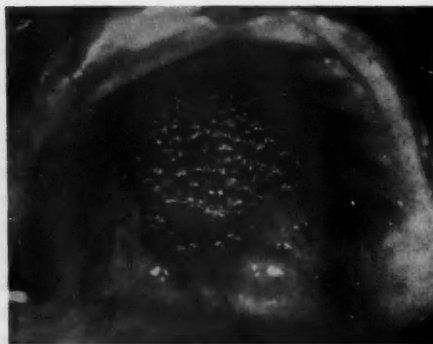

Fig. 1.   Fig. 2.

Fig. 1. Papillary hyperplasia of the palate of a 50 year old man who had been wearing a denture with a "relief area" over the region of growth.

Fig. 2. Biopsy specimen from papillary hyperplasia seen in Figure 1. The papilla occupies most of photograph. Deep within the tissue elongated pegs were found in the connective tissue and dyskeratosis (abnormal maturation) of epithelium was evident.

ever possible, is to eliminate the irritants which, in most cases, are coal tar products. If no areas of redness are observed within the field of whiteness and if no induration, verrucous proliferations or ulcerations are present it is probably clinically sound to eliminate the irritant and keep the lesion under strict observation, and not to resort to surgery unless adverse changes appear or the lesion progresses after removal of the irritation. In any but the mildest, clinically hyperkeratotic lesions, biopsy is indicated. Of course, it is often advantageous to remove small lesions in toto and concurrently to eliminate irritants. The clinician must remain cautious, suspecting and alert but need not be stampeded into unnecessary surgery or into over-stressing his patient.

Another lesion that may be "precancerous" is the papillary hyperplasia sometimes observed in the palate under dentures (Fig. 1). This change is quite different from the epulis fissuratum described by Toto (p. 687) and Gorlin (p. 663). The papillary lesion is red, sessile, and has a raspberry-like surface. It is observed primarily in regions beneath the relief area of upper dentures. Although we have observed

it almost exclusively under acrylic resin dentures, we now see so few dentures of other materials that we must not indict the material on this basis alone. Histologically the lesion usually shows dyskeratosis and, while only occasionally frankly malignant in our experience, it is classified as pseudo-epitheliomatous hyperplasia—resembling malignant change but lacking invasive features characteristic of frank carcinoma (Fig. 2). The same discussion that applies to leukoplakia as a "precancerous" lesion applies to papillary hyperplasia. It should be treated by removing the irritating denture and, unless regression occurs after elimination of the offending denture, by surgical excision. In patients with this lesion we seldom can wait for complete regression before replacing the denture and we therefore resort to surgery. Hobaek[2] considered 21 of 55 carcinomas of the hard palate to be caused by denture irritation, evidence that makes this lesion more suspect than white lesions.

THE DENTIST'S OBLIGATION

The earlier that any carcinoma is discovered, all other factors being equal, the greater the chance of preventing death or "living death"[7] from that cause. The dentist must suspect the possibility of neoplastic disease in any lesion he treats within the oral cavity except for the strictly dental lesions. He must complete his diagnosis in all cases or, if this is impossible, refer the patient to someone qualified to make the diagnosis. This does not mean a mere suggestion that the patient see his "doctor" but that a definite referral be made to a competent person, dentist or physician, with special knowledge of oral disease.

Lesions discovered early (Fig. 3) are less likely to have metastasized than those referred for treatment late in the disease (Fig. 4). Once a carcinoma has been discovered it is most important that the patient be referred for proper and adequate treatment. A cancer clinic where competent personnel usually are available is the ideal referral point for the cancer patient. The dentist who discovers a malignancy has a real responsibility to get the patient under competent care. The surgeon or radiation therapist can treat small, localized lesions with less destruction of tissue and much more probability of cure than he can large, fulminated lesions. The dentist may be a valuable aid as a consultant during surgery to advise on extent of resection as it affects prosthesis, in removing teeth before radiation therapy,[9] and in reconstructing beyond the limits of plastic surgery, as outlined by Heintz (p. 743) and Bitonte (p. 749). Proper removal of teeth may prevent destructive postradiation changes that may make survival a "living death." Prosthesis may rehabilitate the patient for whom necessarily extensive surgery has made social contacts unbearable.

The responsibilities of the dentist in diagnosis are broad. He is the member of the health services team who is most frequently consulted by patients.

The dentist can do much to reduce the unnecessary death toll from oral cancer by preventive measures, and by aiding in early diagnosis. His responsibilities continue as a consultant to the surgeon or radia-
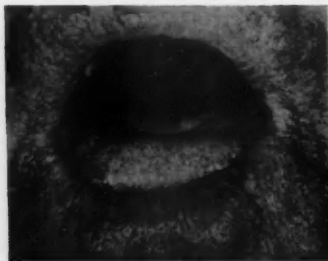

Fig. 3.   Fig. 4.

Fig. 3. Early squamous cell carcinoma on the lip of a 65 year old man. This neoplasm had not yet involved lymph nodes and the prognosis is favorable. (From Thoma and Robinson: Oral and Dental Diagnosis. Philadelphia, W. B. Saunders Co., p. 151.)

Fig. 4. Advanced carcinoma of the lip in a relatively young patient. Note that the regional lymphatics are enlarged. The prognosis is grave. (From the Ellis Fischel State Cancer Hospital, Mo.)

tion therapist and as a principal in prosthetic reconstruction after necessarily destructive therapy.

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Tumors of the Salivary Glands

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The numerous and diverse classifications, theories of origin, and behavior patterns reported for many of the tumors that involve the salivary glands attest to the difficulties encountered in the study of tumors of this group. Further sources of confusion are the multiplicity of names for some of these tumors and the application of the same term to lesions that are entirely dissimilar and distinct.

TABLE 1. *The Salivary Glands*

GLAND OR GLANDS	TYPE OF SECRETORY CELL
Parotid	Serous
Submaxillary	Mainly serous with few mucous
Sublingual	Mainly mucous with few serous
Accessory sublingual	Mixed, mainly mucous
Of the lip	Mixed, mainly mucous
Of the cheek	Mixed, mainly mucous
Glossopalatine	Pure mucous
Anterior lingual	Mixed
Of Von Ebner (associated with circumvallate papillae)	Serous
Of the root of the tongue	Mucous
Of the posterior half of the hard palate	Mucous
Of the soft palate	Mucous

The salivary glands originate as epithelial invaginations from the primitive oral cavity, and, by repeated branching, form the duct system of each gland. The acinar portions are formed later by differentiation of the ductal epithelium. In a regenerating gland the ductal cells provide the source from which the differentiation of the acini proceeds and in obstructive disease of the salivary glands the duct system persists long after the acinar cells have degenerated and disappeared. These facts indicate that the ductal epithelium is the

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most resistant and multipotent component of the glandular parenchyma. Therefore it is believed that the majority of the salivary gland tumors arise from the ductal epithelium.

Besides the three major glands, minor salivary glands occur almost everywhere in the oral cavity (Table 1).

In addition, ectopic salivary gland tissue may occur in the neck and very rarely within the mandible, and under unusual circumstances it may be the site of neoplasia.

The salivary gland tumors may be classified as follows:

EPITHELIAL

Benign	*Malignant*
Acinar and ductal adenoma	Malignant mixed tumor
Papillary cystadenoma lymphomatosum	Mucoepidermoid carcinoma
Oncocytoma	Adenocystic carcinoma
Mixed tumor	Acinic cell carcinoma
	Adenocarcinoma

MESENCHYMAL

Benign	*Malignant*
Fibroma	Lymphoma
Lipoma	Fibrosarcoma
Hemangioma and lymphangioma	
Schwannoma, neurofibroma, neuroma	
Juvenile hemangioma	

EPITHELIAL TUMORS

Ductal and Acinar Adenomas

True acinar and ductal (canalicular) adenomas of the salivary glands are rare. As compared with other adenomas of the salivary glands they are usually smaller, better encapsulated, and less aggressive. Although they may occur anywhere in the major or minor salivary glands, the general impression is that they are more common in the minor glands. These tumors are usually seen after the third decade of life.

Histologically, the ductal adenomas appear as well encapsulated nodules of various sizes. Their parenchyma consists of a monotonous succession of ducts lined by cuboidal or columnar cells, which closely resembles the normal ductal epithelium (Fig. 1). The lumen of these ductal structures either is empty or contains homogeneous eosinophilic material. The acinar adenoma resembles that of the ductal type in all respects except that its parenchyma is made up of islands and clusters of acinar cells of mucous or serous types.
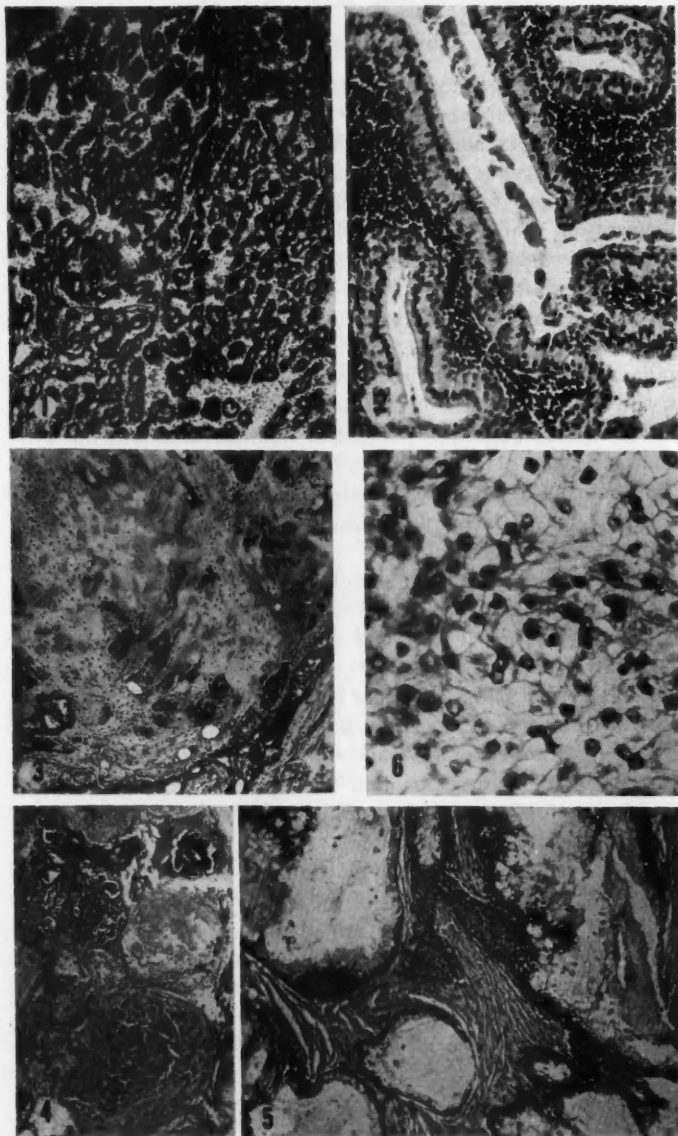
Fig. 1. Tubular adenoma. ×55.
Fig. 2. Papillary cystadenoma lymphomatosum. ×125.
Fig. 3. Pseudocartilaginous areas in a mixed tumor. ×55.
Fig. 4. Pleomorphic pattern in a mixed tumor. ×4.
Fig. 5. Mucoepidermoid carcinoma. ×55.
Fig. 6. Clear cells in an acinic cell carcinoma. ×400.

Papillary Cystadenoma Lymphomatosum

The papillary cystadenoma lymphomatosum, or Warthin's tumor, has aroused considerable controversy regarding its histogenesis, site of origin and proper nomenclature. This tumor is known by at least sixteen different names and at least six different theories of origin have been claimed for it. The belief that papillary cystadenoma lymphomatosum is solely a tumor of the parotid gland is deeply rooted, but recent investigation has shown that although it usually involves the parotid gland, it can occur in the neck, in a submaxillary gland, or in any area where salivary gland tissue is closely associated with lymph nodes.

Papillary cystadenoma lymphomatosum is benign, usually occurs in males, and is seen most frequently in the fifth or sixth decade of life. It usually has a long history of slow growth unaccompanied by symptoms of facial nerve involvement. The tumor, when superficial, may be fluctuant. Its recurrence rate is not as high as that of the mixed tumor, but, if incompletely excised, it does recur. It does not undergo malignant degeneration.

Grossly the tumor appears encapsulated and its cut surface usually shows numerous small cystic spaces which exude a thick, sticky fluid. Histologically (Fig. 2) it is a multicystic lesion in which the cyst walls are thrown into numerous folds and papillae. The walls are lined by a double layer of cells. The inner layer is columnar, deeply eosinophilic, with distally placed, deeply basophilic nuclei, while the cells of the basal layer are cuboidal, also deeply eosinophilic, and with large vesicular nuclei. The cysts contain homogeneous eosinophilic material. This papillary cystic pattern lies in a background of dense, diffuse and follicular lymphoid tissue.

In addition to the papillary cystadenoma lymphomatosum, there are other benign epithelial tumors of the salivary glands in which the epithelial and lymphoid elements are closely associated. These are the adenoma lymphomatosum and the pleomorphic adenoma lymphomatosum.

Oncocytoma

The oncocytoma, or oxyphil cell adenoma, is a benign epithelial tumor which supposedly is peculiar to the parotid, although extraparotid oncocytomas do occur. These tumors are believed to arise from the so-called oncocytes described by Hamperl, very large eosinophilic cells with clear outlines and small dark or vesicular nuclei. They occur in persons more than 50 years of age, not only in

the parotid but also in many other parts of the body. Thus the oncocytoma is a tumor of old age. Its growth is slow and persistent, usually unattended by pain or involvement of the facial nerve. Since the tumor is circumscribed and well encapsulated, local removal should be curative, though recurrences are possible. It does not undergo malignant change and does not metastasize.

Grossly the oncocytoma is invariably circumscribed and encapsulated and its cut surface is not cystic. Microscopically, the lesion is composed exclusively of oncocytes in solid sheets, clusters, columns, and cords.

Mixed Tumor

The most common tumor of the salivary glands, particularly the major glands, is the so-called mixed tumor, or pleomorphic adenoma. Of the major salivary glands the parotid is the site of about 90 per cent of these tumors; only 8 to 9 per cent involve the submaxillary gland, and the rest occur in the sublingual glands. The term "mixed tumor" was applied to this lesion because it was believed that it originated from both epithelial and mesenchymal elements; however, it is now generally accepted that the tumor is exclusively epithelial.

Mixed tumors usually occur in the third and fourth decades and have a slightly higher predilection for women than men. The tumors, when primary, are usually uninodular and grow very slowly. They rarely involve the facial nerve. Recurrence rates ranging from 5 to 30 per cent have been reported, reflecting the thoroughness with which the primary lesion was excised. When the tumor recurs it often is multilobulated and multinodular and the chance of subsequent recurrence is enhanced owing to the mechanical difficulties encountered in secondary removal. Although the mixed tumor does not metastasize, it is locally destructive. Malignant degeneration in these tumors, although rare, does occur; the interruption of a long history of slow growth by a sudden spurt of proliferation, accompanied by ulceration or facial paralysis, or both, is a highly ominous sign.

Clinically, these tumors, if superficial, are circumscribed, smooth and movable; if deep, they are difficult to palpate. Gross specimens are uninodular or multinodular, encapsulated, and on cut surface have a variegated appearance. Cystic, cartilaginous, hemorrhagic, mucinous and solid areas of various shapes and sizes may be seen. Histologically, the picture is even more diverse (Figs. 3 and 4). Polyhedral, cuboidal, columnar, fusiform and stellate cells form sheets,

islands, clusters and duct-like structures, or any other pattern imaginable. Homogeneous eosinophilic or basophilic material can be seen within or without the duct-like structures. In some places squamous metaplasia and keratin formation may occur. Very frequently the epithelial secretions, which are mucicarmine positive, accumulate in islands of various sizes and entrap the epithelial cells to give a superficial resemblance to cartilage. This is the feature which is the source of the erroneous impression that these tumors are "mixed," that is, both epithelial and mesenchymal in origin. Areas of hyalinization are not uncommon in mixed tumors. All these patterns or various combinations of them may be seen in a single tumor or in different tumors.

There are a few salivary gland tumors in which one of the cell types described may form a predominant part, if not the whole. These cellular elements are usually arranged in very dense sheets, but the cytologic features of malignancy are lacking. The cells are of uniform size and staining qualities and do not contain excessive numbers of mitoses. These tumors are referred to as cellular mixed tumors on the yet unproven assumption that they are perhaps more aggressive and more likely to recur than the usual mixed tumor.

Malignant Mixed Tumors. A few salivary gland tumors after a long, slow course have a sudden spurt of growth, and histologically show areas of frank carcinoma as well as areas indistinguishable from the ordinary mixed tumor. Apparently such tumors represent malignant transformation in a pre-existing benign lesion and are referred to as malignant mixed tumors. Clinically, a sudden spurt of growth in a tumor of long duration, particularly when associated with involvement of the facial nerve and ulceration of the overlying skin, is a sign which forecasts a grave outcome. Malignant mixed tumors metastasize and kill.

Mucoepidermoid Carcinoma

Until recently the mucoepidermoid tumor was believed to occur in benign and malignant forms. Now, however, the consensus is that these tumors are all malignant (although in varying degree). Of the major salivary glands the parotid is the most frequent site of such tumors; of the minor glands those of the posterior part of the palate are most likely to be involved. The tumor usually occurs in persons between the ages of 30 and 50 years and appears to be commoner in women. Clinical features vary widely in relation to the grade of malignancy. Tumors of low-grade malignancy are of long duration and

do not cause facial nerve symptoms or ulceration, whereas tumors of high-grade malignancy are characterized by rapid growth, facial nerve involvement, ulceration of overlying skin and mucous membrane, and metastases.

Grossly, the mucoepidermoid carcinoma is cystic or solid, encapsulated or diffuse. When cystic, the cysts exude a mucoid fluid. In the tumor of high-grade malignancy the cystic features are not particularly prominent, but areas of necrosis are common.

Microscopically, the mucoepidermoid tumor is characterized by the presence of cells of three types: epidermoid, mucous, and so-called intermediate (Fig. 5). The last named is considered to be the parent cell of the other two types. The predominant feature of these tumors is the variety of cystic spaces, which range from large folded cystic cavities to microcysts. The cystic spaces are lined by mucous cells of various shapes and sizes and also cells of the squamoid and intermediate types. The spaces contain mucin-positive material. In many areas the cystic feature is absent and sheets and islands of the three cell types in various combinations predominate.

Clear-cut differentiation between the mucoepidermoid tumor of low-grade and high-grade malignancy is difficult. It has been suggested that cysts are a common feature of the tumors of low-grade malignancy and the predominant cellular elements are the mucous and epidermoid cells. In the lesions of high-grade malignancy, on the other hand, cysts are uncommon and the epidermoid and intermediate cell types predominate.

It must be emphasized that there are other lesions of the salivary glands in which mucous production may be associated with both cyst formation and squamous metaplasia of the glandular epithelium. These lesions often are mucous cysts or obstructive adenitis and should not be mistaken for mucoepidermoid tumors.

Adenocystic Carcinoma

The adenocystic carcinoma, a malignant tumor, goes by a variety of names, among which are basaloid mixed tumor, epithelioma adenoides cysticum, and cylindroma. The last mentioned, though popular, is a very poor term, since it is also used for the so-called turban tumor, a benign lesion of the skin which occurs on the face and scalp. The adenocystic carcinoma occurs most commonly in the palate, the parotid and the submaxillary glands. These tumors occur most frequently in persons past 40 years of age and exhibit no predilection for members of either sex. They grow relatively slowly, but are persistent and recur repeatedly. After multiple recurrences they

metastasize to regional lymph nodes, lungs, brain, bone and other structures. Because of their aggressive nature, however, they can kill by local extension; for example, a tumor of the palate progressively destroys bone and invades the cranium. The clinical appearance of the lesion varies. Primary lesions are usually uninodular, while the recurrences are predominantly multinodular. Lesions of the palatal glands may be associated with loosening of teeth. Roentgenograms in such cases show bone destruction, and if teeth are extracted the sockets fail to heal. When the parotid gland is the site of adenocystic carcinoma, symptoms of involvement of the facial nerve occur in some cases and, in spite of the slow growth of the tumors, are a danger signal. The gross specimen resembles that of a mixed tumor except that encapsulation is usually incomplete or absent.

Microscopically, the predominant and almost exclusive cellular element is a small epithelial cell with scanty cytoplasm and a small dark nucleus, which resembles a basal cell of mucous membrane. Because of this feature it is sometimes called a basaloid mixed tumor. The epithelial cells are arranged in tubes, islands, columns and acini. The tubes and acini either contain an eosinophilic or a basophilic material or are empty. In areas where the empty tubes and acini predominate, the "Swiss cheese" appearance is characteristic. The stroma is moderate or scanty in amount and is arranged in columns or cylinders or, as is usually the case, is indifferent. One of the peculiar features of the lesion is its invasion of and extension into the perineural lymphatics.

Acinic Cell Carcinoma

Acinic cell carcinoma is a malignant tumor which occurs mainly in the parotid and has a higher incidence in women than in men. Clinically, it has many features of the mixed tumor for which it is usually mistaken. When the site of such a tumor is the parotid gland, paralysis of the facial nerve, although not common, may occur. The tumors are likely to recur after excision and may metastasize to regional nodes or distant areas.

Histologically, the acinic cell carcinoma is characterized by sheets and islands of large polyhedral cells, supported by delicate or dense connective tissue stroma. The cells either have deeply basophilic, coarse granules in their cytoplasm and resemble the serous cells of the normal gland or have clear cytoplasm (Fig. 6). Tumors consisting exclusively of one or the other cell type may be seen, or a tumor may show cells of both types. When the clear cells form the prominent part or the whole of a tumor, a superficial resemblance to the hyper-

nephroma is observed, and for this reason some of these neoplasms have been called "hypernephroid" or "parathyroid-like tumors."

Adenocarcinoma

In addition to the tumors described, other highly malignant neoplasms of the salivary glands occur. They are not common, but because of their behavior early diagnosis is imperative. Such tumors are seen most frequently in the parotid. Their history is one of rapid growth, fixation to tissues, involvement of the facial nerve, and, in some cases, ulceration of overlying skin or mucous membranes occurs. Metastases are seen earlier than in salivary gland tumors of other types. The histologic appearance of these tumors is varied and, accordingly, different types may be recognized. In essence, however, the most striking features are anaplasia, mitoses, pleomorphism and lack of a consistent pattern of cellular arrangement.

MESENCHYMAL TUMORS

Tumors of mesenchymal origin are rare in the salivary glands. Fibromas, lipomas, neurogenic tumors and hemangiomas are seen occasionally and are identical to their counterparts elsewhere. The tumor of greatest interest in this group is the so-called juvenile hemangioma. It is seen in infants and children and most frequently involves the parotid gland. Clinically the gland is enlarged but painless. The tumor has varying rates of growth and, if sudden canalization of the vascular channels should occur, it might show an impressive increase in size. Microscopically, the lobular pattern of the gland is unchanged. In the lobules, however, numerous endothelium-lined blood vessels are seen among the acini. The interlobular connective tissue apparently is not involved by the tumor. In all probability the juvenile hemangioma represents a malformation rather than a true neoplasm.

Malignant mesenchymal tumors of salivary glands are extremely rare.

RELATIVE INCIDENCE OF SALIVARY GLAND TUMORS

A review of more than 2300 tumors of the salivary glands reveals the following incidence of the various types: mixed tumor, 1726; adenocarcinoma, 165; papillary cystadenoma lymphomatosum, 160; mucoepidermoid carcinoma, 100; adenocystic carcinoma, 45; malignant epithelial tumors, unclassified, 37; lymphangiomas and hemangiomas, 33; oncocytomas, 20; neurogenic benign tumors, 17; adenoma, 10; acinic cell carcinoma, 8; hamartoma, 7; malignant mixed tumor, 5; reticulum cell sarcoma, 5; myxoma, 2; lipoma, 1; fibrosarcoma, 1.

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Tumors of the Lips

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The lips are composed of skin, mucous membrane and muscle tissue. Between the cutaneous and mucosal surfaces of the lips lies a transitional zone which is devoid of mucous and sebaceous glands. This zone, the vermilion border of the lip, is prone to drying and needs to be moistened frequently by the tongue, and is the most frequent site of epithelial neoplasms of the lips.

TABLE 1. *Tumors of the Lip*

TISSUE AFFECTED	BENIGN	MALIGNANT
Covering epithelium	Papilloma Verruca vulgaris	Squamous cell carcinoma
Salivary glandular epithelium	Adenoma Mixed tumor	Malignant mixed tumor Adenocarcinoma Mucoepidermoid carcinoma
Blood and lymph vessels	Capillary hemangioma Cavernous hemangioma Lymphangioma	Malignant hemangioendothelioma
Connective tissue	Irritation fibroma Fibroma Lipoma	Fibrosarcoma
Nerves and nerve sheath	Neuroma Neurofibroma Schwannoma	Malignant schwannoma
Muscle	Leiomyoma Rhabdomyoma	Leiomyosarcoma Rhabdomyosarcoma
Miscellaneous	Granular cell myoblastoma Nevus Juvenile melanoma Hamartoma and choristoma	Melanoma

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The skin of the lips contains the usual adnexa. In the submucosa of the mucosal side of the lip there are numerous salivary glands of mixed type whose ducts open independently onto the mucosal surface.

The bulk of the lips is formed by the striated musculature. In addition to the muscles, the covering epithelia, the hair follicles, and the sebaceous, sweat, mucous and serous glands, the lips contain blood vessels, lymph vessels, nerves and fibrous connective tissue. The lymph vessels from the lower lip drain into the submental and submandibular lymph nodes, those of the upper lip into the submandibular and the superficial parotid nodes.

Since a portion of the lips is covered by skin, it is obvious that the cutaneous surface may be the site of all neoplasms seen in the skin elsewhere. Tumors arising from the sebaceous glands, hair follicles and sweat glands form a distinct group and will not be included in this discussion. It need only be remembered that these lesions may occur within the anatomic boundary of the upper or lower lips.

TUMORS OF COVERING EPITHELIUM

Papilloma is a benign epithelial tumor which can occur at any age, usually on the inner surface of the lip. Clinically it presents a cauliflower-like appearance and is attached to the mucous surface by a slender stalk (Fig. 1).

Microscopically, one sees numerous finger-like projections covered by keratinized or parakeratotic stratified squamous epithelium. These projections carry narrow, highly vascularized connective tissue cores, all connected to the stalk of the lesion. The epithelium of these lesions does not show dyskeratosis, and the connective tissue is either free of inflammatory cells or has but a minimal number.

Verruca vulgaris is a wart-like lesion which, although not a true neoplasm, resembles a papilloma. It is caused by a virus and develops much more rapidly than a papilloma. In the oral cavity it occurs most frequently on the labial mucosa where it may easily be traumatized. Recurrence is not unusual. Verrucae may occur singly or in groups, are raised above the surrounding mucosa, and have a horny appearance.

Microscopically, verruca vulgaris is papillary with a covering of parakeratotic stratified squamous epithelium. The crests of the epithelial papillae are covered by a layer of parakeratotic cells with keratin between the papillae. Intranuclear inclusion bodies have been described in the keratinized layer. The epithelial papillae contain cores
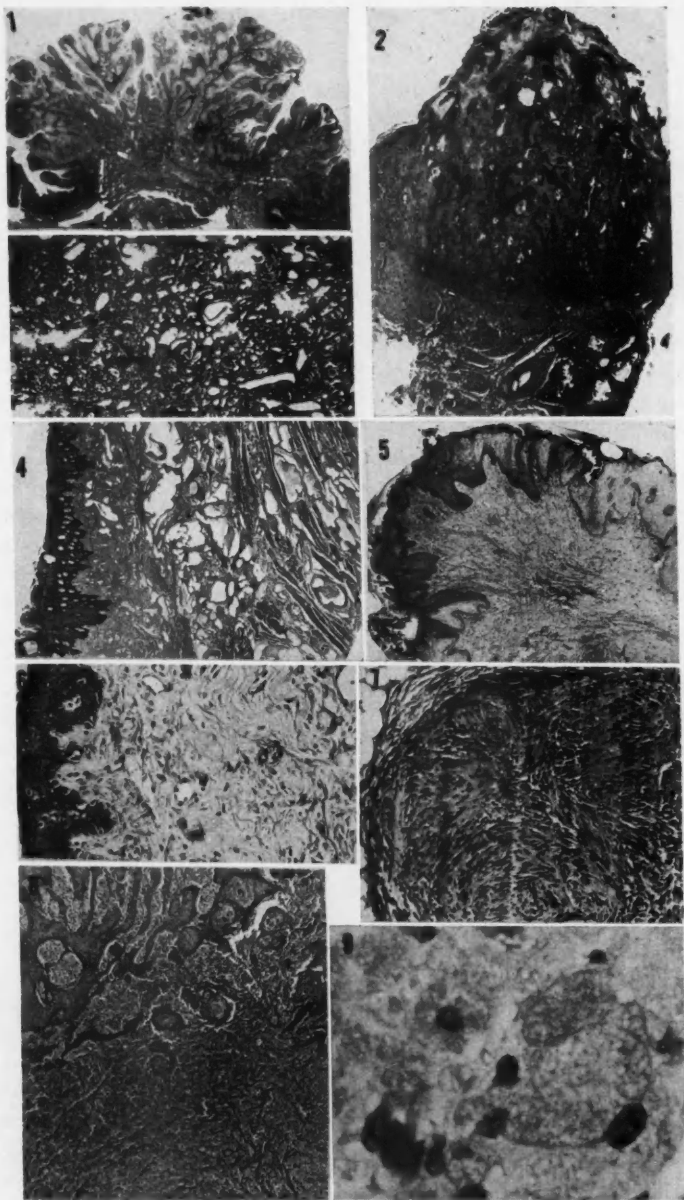
Fig. 1. Papilloma. ×5.
Fig. 2. Squamous cell carcinoma. ×5.
Fig. 3. Juvenile hemangioma. ×32.
Fig. 4. Lymphangioma. ×9.
Fig. 5. Irritation fibroma. ×12.
Fig. 6. Irritation fibroma. ×40.
Fig. 7. Schwannoma, ×65.
Fig. 8. Granular cell myoblastoma. ×9.
Fig. 9. Granular cell myoblastoma. ×200.

of connective tissue in which mild inflammatory exudate can some-
times be seen. At the verrucal margins the epithelial ridges charac-
teristically are bent inward and appear to point radially toward the
center.

Squamous Cell Carcinoma. The significance of certain lesions re-
lated to squamous cell carcinoma and terms used to identify them
must be fully understood. It is generally believed that *leukoplakia*
is a premalignant lesion, but the term leukoplakia is sometimes ap-
plied rather loosely to any white patch on the oral mucous membrane.
A variety of oral lesions are known to appear clinically as white
patches, and certainly few, if any, are premalignant. An accurate tis-
sue analysis of most such areas is therefore essential to diagnosis.
Two such lesions are of special importance. One is a white patch of
the oral mucosa, the covering epithelium of which has a thick layer
of keratin although otherwise the cellular detail and structure remain
unaltered. Dyskeratosis is absent. Such a lesion has been called
pachyderma oris, or *hyperkeratosis,* and probably has no connection
with malignant disease. The second is a white patch which histolog-
ically shows hyperkeratosis or parakeratosis and, in addition, evidence
of disturbed maturation in the epithelial cells. "Dyskeratosis" denotes
such abnormal maturation of cells. Altered cellular arrangement, nu-
clear detail, mitoses or other abnormalities are indicative of biologic
disturbance. The basement membrane is intact, and inflammatory
cells may or may not be seen in the subepithelial connective tissue.
Such a lesion is called *leukoplakia.* The importance of distinguishing
between the two lies in the belief that some of the lesions which
show dyskeratosis may become malignant. This is not to say that all
leukoplakias are carcinomas in the making, but only that some carci-
nomas arise in pre-existing leukoplakias. Therefore, the implications
with regard to treatment are obvious.

Carcinoma in situ, a lesion closely related to leukoplakia and car-
cinoma, is characterized by the presence of pronounced dyskeratosis
in the covering epithelium. Cellular abnormalities are seen in all
layers, but the basement membrane is intact and invasion of the
corium is not demonstrated. These lesions obviously must be treated
by careful and complete removal.

Squamous cell carcinoma is the most common malignant oral
tumor, constituting more than 90 per cent of all malignant tumors in
that region. It occurs more frequently on the lips than intraorally,
and of all such tumors on the lips, 95 per cent occur on the lower
lip and only 5 per cent on the upper lip. Although the incidence
of the tumor is highest after the fourth decade, a large number

of squamous cell carcinomas of the lip have been reported in a younger age group. They are more frequent in whites than in Negroes and their incidence in the male is fourteen times greater than in the female.

Bernier and Clark[2] found that clinically the tumor is seen initially in a wide variety of forms, with the relative frequency shown in Table 2.

Microscopically, squamous cell carcinoma violates the basement membrane and lays down cords, islands and sheets of abnormal squa-

TABLE 2. *Initial Clinical Features of Carcinoma of Lip*

CLINICAL FEATURE	NUMBER	PER CENT
Ulcer	227	28.6
Wart	141	17.8
Sore	130	16.4
Scab	87	11.0
Blister	63	8.0
Fissure, crack	62	8.0
Scale, scaly	23	3.0
Crusting	16	2.0
Sunburn	11	1.3
Tumor	9	1.1
Knot	9	1.1
Trauma, residual of	5	0.6
Other (plaque, infection, keratosis, cyst)	9	1.1
Total	792	100.0

mous epithelium in the subepithelial tissues (Fig. 2). The invading epithelium exhibits various features that are summarized by the term dyskeratosis. The cellular changes cover a wide range, and carcinomas of well differentiated to highly anaplastic types are recognized. The well differentiated tumors often show abundant keratin pearl formation and epithelium is easily recognizable. In the anaplastic forms, on the other hand, it may be difficult to interpret the tumor as epithelial. The significance of recognizing well differentiated and poorly differentiated types lies in the fact that usually the former grow more slowly than the latter. These tumors were graded by Broders according to the percentage of abnormal cells. Tumors with less than 25 per cent were classified as grade I, those with 25 to 50 per cent as grade II, those with 50 to 75 per cent as grade III, and those with 75 to 100 per cent as grade IV. However, it should be realized that in evaluating a tumor its size and duration are just as important as its grade. An early grade IV carcinoma which can be

excised may have a better prognosis than a far advanced tumor of a lower grade.

Of all squamous cell carcinomas of the oral cavity, those of the lip have the best prognosis, partly because they come to the attention of the patient quickly, and partly because they usually are well differentiated. Squamous cell carcinoma of the lip spreads via the lymphatics; tumors of the lower lip usually metastasize to the submental and submaxillary lymph nodes, those of the upper lip to the submaxillary and periparotid nodes. Carcinoma of the lip is best treated by surgical excision.

TUMORS OF SALIVARY GLANDULAR EPITHELIUM

Adenoma. Tumors of salivary gland origin in the lip are identical to their counterparts elsewhere. Simple ductal and acinar adenomas in the lip are rare. When they do occur, they are likely to appear as small circumscribed nodules which are diagnosed clinically as fibromas or mucoceles. Histologically, these lesions show a monotonous arrangement of ducts or acini.

Mixed Tumor. Of the salivary gland tumors that arise in the lip, the mixed tumor is the most common and is usually seen in patients past the third decade. It occurs much more frequently in the upper than the lower lip, possibly because of the more complicated development of the upper lip. The morphologic features, natural history and treatment of these tumors are identical to those of similar tumors elsewhere.

Malignant Mixed Tumor. A mixed tumor may undergo malignant degeneration. These tumors are discussed in detail in the article on tumors of the salivary glands.

Adenocarcinoma. Various types of adenocarcinoma of salivary gland origin occur in the lips.

Mucoepidermoid Carcinoma. In the lips this tumor does not differ from its counterparts elsewhere.

TUMORS OF BLOOD AND LYMPH VESSELS

Hemangioma. In the lips, as in other locations, a tumor or a tumor-like mass consisting of blood vessels may be one of three types: (1) a malformation or developmental anomaly; (2) an abnormal tissue response to irritation or infective agent; (3) a true tumor.

1. Developmental defects consisting of blood vessels, lymph vessels or both, are well known. Cirsoid aneurysms, the skin lesions of

Sturge-Kalischer-Weber syndrome and Lindau-von Hippel disease no doubt belong to this group, and possibly the so-called juvenile hemangioma in very young children or infants. When in the oral region, the latter most often involves the parotid gland or the lips, growing slowly and causing a diffuse enlargement of the lip. Histologically, these lesions are made up of a multitude of small endothelium-lined blood channels which may or may not contain blood (Fig. 3). These capillaries diffusely permeate the tissues of the area. The surface of the growth usually is not ulcerated. Opinions of clinicians regarding the management of these lesions differ considerably. Some prefer to leave them undisturbed on the assumption that with age they regress; others advise radiation or surgical intervention.

2. Tumor-like masses consisting of blood vessels may be seen in lesions which actually are an unusual tissue response to injury. To this group belongs the pyogenic granuloma which may occur on the lip. To distinguish between a pyogenic granuloma and a capillary hemangioma is often difficult. However, in many instances, the presence or absence of inflammatory exudate, the status of the covering epithelium and the duration of the lesion are of considerable value in differential diagnosis.

3. True neoplasms of the blood vessels are seen at all ages. These capillary or cavernous hemangiomas are fairly common on the lips, where they vary from less than a centimeter in diameter to the size of a plum. They may be sharply demarcated or diffuse; their surface may be smooth or lobulated, and their color varies from purple to bright red. If injured, they bleed profusely.

Histologically, the capillary hemangioma is composed of numerous small endothelium-lined channels, while the cavernous type contains large irregular spaces lined by a layer of endothelium. Uncanalized masses of endothelium may become canalized and the lesion may show sudden enlargement.

Lymphangiomas. Circumscribed or diffuse lesions of the lip, consisting predominantly of lymph vessels, are called lymphangiomas (Fig. 4). They occur at any age and may produce considerable enlargement of the lip.

Malignant hemangioendothelioma, or angiosarcoma, the malignant counterpart of the hemangioma, is rare in the lips. It grows rapidly and blood-borne metastasis occurs early.

TUMORS OF CONNECTIVE TISSUE

Irritation fibroma is a common tumor-like growth of the lips. Usually a circumscribed, sessile or pedunculated growth on the mucosal

surface, it is associated with trauma, such as that caused by habitual sucking. These lesions of slow and limited growth occur at any age. Histologically, the major mass of the irritation fibroma consists of densely collagenized tissue in which there are areas of hyalinization (Figs. 5 and 6). The entire lesion is covered by a layer of stratified squamous epithelium which may show parakeratosis or hyperkeratosis. Such lesions are not true neoplasms but represent a hyperplastic response to irritation.

Fibroma. True fibromas of the lip are rare.

Lipoma. Lipoma is a slowly growing benign tumor which occasionally attains a large size. It is generally superficial and tends to be pedunculated; however, when it is located in deeper tissues, it produces a smooth enlargement. Microscopically, the tumor is composed of mature fat cells.

Fibrosarcoma. Fibrosarcomas are rare in the lip. When they are seen they are similar to those that occur in the other soft tissues.

TUMORS OF NERVES AND NERVE SHEATH

Neuroma. Neuroma, although not a true tumor, is a tumor-like proliferation which occasionally develops after trauma, including surgical procedures. It consists of nerve fibers, Schwann cells and fibroblasts. These tumor-like growths may occur in the lips, and when painful, they should be excised.

Neurofibroma and Schwannoma. These benign tumors of nerve sheath origin are sometimes described as distinct entities, but for the purposes of the present discussion they will be considered identical. In the oral region they are seen most frequently in the tongue but also in the lips, usually as small submucosal nodules that are round or fusiform and movable, and sometimes painful. Microscopically, they consist of tissue of two types referred to as Antoni A and B. Antoni type A (Fig. 7) is characterized by solid masses of Schwann cells in bundles and cords, or arranged so as to produce palisading of nuclei and nucleus-free areas (Verocay bodies). In Antoni type B tissue a loose, almost myxomatous arrangement of cells is associated with the formation of microcysts. These lesions do not recur after excision. However, neurofibromas and schwannomas associated with multiple neurofibromatosis not only recur but may undergo malignant transformation.

Malignant schwannoma, or neurofibrosarcoma, is a malignant tumor of neurogenic origin. It is rare in the lip.

TUMORS OF MUSCLE

Leiomyoma, rhabdomyoma, leiomyosarcoma and rhabdomyosarcoma. Benign and malignant tumors of muscle tissue origin are rare in the lips. Recently a variety of rhabdomyosarcoma, referred to as the embryonal rhabdomyosarcoma, has been described. This highly malignant, rapidly growing tumor of immature rhabdomyoblasts usually occurs in children.

MISCELLANEOUS TUMORS

Granular Cell Myoblastoma. Granular cell myoblastoma is a benign tumor or tumor-like growth of unknown nature. In the oral region it is most commonly observed in the tongue and the lips. It may occur at any age and is usually asymptomatic. Clinically it is seen as a painless swelling of varying size and duration. The histogenesis of this lesion is a matter of controversy. Histologically the tumor consists mainly of very large multisided cells with granular cytoplasm and small rounded nuclei (Figs. 8 and 9). These cells are arranged in sheets and are supported by a delicate reticular stroma. The epithelium covering these lesions shows pseudo-epitheliomatous hyperplasia which may be mistaken for epidermoid carcinoma (Fig. 8). Local excision is curative.

Nevi. Junctional, intradermal and compound nevi are often seen in the lips. In this location they do not differ from their counterparts elsewhere in the body.

Melanoma. Malignant melanomas are rare in the lips. Like similar tumors elsewhere they are highly malignant. Juvenile melanomas, on the other hand, resemble malignant melanomas histologically but behave as benign lesions. They occur exclusively in children.

Hamartomas and Choristomas. Hamartomas and choristomas consisting of an abnormal amount or mixture of various tissue elements are sometimes seen in the lips. They are tumor-like enlargements made up of cartilage, bone, muscle, blood vessels, nerves or other tissues, alone or in various combinations. They grow slowly or are quiescent. Histologically, they are composed of tissues which are abnormal only in amount or location. They do not recur after excision.

RELATIVE INCIDENCE OF TUMORS OF THE LIP

A review of more than 2100 cases of tumors of the lip shows the following relative incidence of various types: epidermoid carcinomas, 961; nevi (intradermal, compound, junctional), 356; papillomas, 302; hemangiomas and pyogenic

granulomas, 236; fibromas, 124; mixed tumors (salivary gland), 103; lymphangiomas, 57; neurofibromas, 32; adenomas (salivary gland), 15; myoblastomas, 12; adenocarcinomas, 10; melanomas, 5; fibrosarcomas, 4.

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Tumors of the Tongue and the Floor of the Mouth

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In this article the tumors of the tongue and floor of the mouth are treated together to avoid needless repetition. Two of the photographs illustrate lesions in locations other than the area under discussion. They are used because they illustrate the tumors well and because the lesion appears the same in the tongue and floor of the mouth.

PAPILLOMA

Etiology and Clinical Features. Papillomas are benign epithelial tumors which occur as an overgrowth of surface epithelium. Trauma, infection, metabolic disturbances and viral origin have been suggested as secondary etiologic factors. The lesions may be single or multiple and vary considerably in size. Papillomas are cauliflower-like in appearance, projecting well above the adjacent tissue in a branching fashion. They are attached by a pedicle or broad base and may be firm or soft on palpation. Lesions in the tongue are often diffuse and involve large areas. Because they are frequently keratotic they appear lighter in color than the surrounding tissues. Ulceration with resultant inflammation produces pain.

Treatment and Histopathology. The treatment of choice is simple surgical excision. The lesion does not tend to recur. Histologically the papilloma is composed predominantly of stratified squamous epithelium which projects above the neighboring mucosa in a tree-like pattern and is supported by a fibrous connective tissue core (Fig. 1). The latter is prominently vascularized and extends into each branching. The epithelium is acanthotic with a well defined basal cell layer. Keratosis may be seen.

FIBROMA

Etiology and Clinical Features. Fibromas originate from the deep layers of mucosa. They may result from trauma, in which case they are referred to as irritation fibromas, or they occur without an obvious cause and are referred to as fibromas. They are benign, well encap-

Fig. 1.   Fig. 2.

Fig. 3.   Fig. 4.

Fig. 1. Papilloma. Proliferating epithelium forming numerous projections, each of which is supported by a small, vascular, fibrous connective tissue core.

Fig. 2. Fibroma. Submucosa is composed of fibrous connective tissue surmounted by stratified squamous epithelium. A narrow pedicle is noted.

Fig. 3. Neurofibroma. A smooth, large, glistening swelling in floor of mouth. It is firm and slightly red in color. Veined appearance is uncommon.

Fig. 4. Lipofibroma. Soft, freely movable lesion, yellow-white in color. Vascular striations are noted on the surface.

sulated and vary in size from a few millimeters to over 1 cm. in diameter. (See p. 681.) Although attachment by a broad base is not uncommon the majority of lesions in the tongue are pedunculated. Fibromas frequently match the adjacent tissues in color but may vary from pink to dark blue. They are firm or soft, depending upon their histologic makeup. Subjective symptoms are seldom pronounced un-

less there is ulceration or unless the tumor has reached such size that it interferes with mastication or speech.

Treatment and Histopathology. The treatment is complete surgical removal. Recurrence is rare. Microscopically the fibroma is composed of bundles of cellular or collagenous fibrous connective tissue (Fig. 2). Vascularity may be marked. On occasion calcified products are observed in small quantity, depending upon the degree of differentiation of the tumor elements. The covering stratified squamous epithelium varies in thickness and keratosis is present in many cases.

NEUROFIBROMA

Etiology and Clinical Features. The neurofibroma is a true neoplasm that arises from the connective tissue of the nerve sheath. Trauma is believed to play a role as a secondary factory and endocrine dysfunction is suggested as a cause in the multiple form of this disease, referred to as von Recklinghausen's disease or multiple neurofibromatosis. In this latter instance a hereditary background is also generally recognized. Although this lesion is essentially benign, it is unencapsulated and recurrence is not uncommon. A neurofibroma is slow growing and painless. It is associated with the peripheral nerves and appears as a movable mass, 1 to 2 cm. in diameter, in the subcutaneous tissues. The tongue is the most common intraoral location. These lesions in the floor of the mouth attain rather large proportions (Fig. 3). Subjective symptoms include paroxysmal pain, difficulty in hearing and swallowing and disturbed taste sensations, depending upon the size and location of the mass.

Treatment and Histopathology. Surgical excision with a wide margin is indicated. Microscopically the tumor is composed of whorls and interlacing bundles of fibrous connective tissue between which are seen elongated cells with wavy nuclei. Myxomatous degeneration may be present but is not a constant finding.

NEURILEMMOMA (SCHWANNOMA, NEURINOMA)

Etiology and Clinical Features. The neurilemmoma is believed to arise from the sheath of Schwann as a solitary lesion along the route of any of the peripheral, cranial or sympathetic nerves, and it is regarded as benign. Small tumors are relatively solid but the larger ones may undergo cystic degeneration. They occur in any age group, in both sexes and in all races. The tumor grows slowly and is usually symptomless. Pain, however, may result from trauma and ulceration when the lesion is on the tongue, a favorite intraoral location.

Treatment and Histopathology. Conservative surgical excision with a wide margin is the treatment of choice. Recurrence is uncommon. Histologically the neurilemmoma is composed of slender cells with elongated nuclei which tend to arrange in rows with intervening spaces void of nuclei, an arrangement described as "palisading."

MYOBLASTOMA

Etiology and Clinical Features. Although a neurogenic origin has been suggested for this benign tumor the most likely pathogenesis appears to be that of degenerating muscle cells or dysontogenesis of embryonal skeletal muscle. Myoblastomas occur most frequently on the tongue, particularly near the tip, appear at any age and have a predilection for males. They are relatively rare and are found chiefly in a middle age group. Characteristically of slow growth, they are elevated and usually lighter in color than the surrounding mucosa. The majority of the lesions are attached by a broad base but on rare occasions are pedunculated. They are small, well circumscribed and produce no symptoms except pain if and when ulceration occurs.

Treatment and Histopathology. Complete surgical excision is the treatment of choice. Myoblastomas do not tend to recur. Microscopically they are composed of large polyhedral granular acidophilic cells, between 20 and 60 micra in size. The nucleus is small and relatively uniform. The stratified squamous epithelial covering may be relatively normal in appearance or hyperplastic to the point of resembling squamous cell carcinoma. These lesions, however, are benign and must not be interpreted as malignant.

LEIOMYOMA

Etiology and Clinical Findings. The leiomyoma is thought to originate from either the embryonic rests of smooth muscle, from muscle normally in situ or from the smooth muscle of vascular spaces. Its rarity in the oral cavity is explained on the basis of the paucity of smooth muscle in this location. This lesion is slow growing and may attain great size. The tumor is well encapsulated, pedunculated or attached by a broad base, and is usually the approximate color of the neighboring mucosa. Lesions at the base of the tongue may cause symptoms of obstruction and voice changes. Pain is unusual.

Treatment and Histopathology. Treatment consists of surgical removal by a wide or narrow margin, depending upon the benignity or malignancy of the lesion. Histologically the leiomyoma consists of

interlacing bundles of smooth muscle fibers separated by strands of fibrous connective tissue. The individual cells are spindle shaped, containing rod-like nuclei and dense acidophilic cytoplasm.

LIPOFIBROMA

Etiology and Clinical Features. The lipofibroma is a benign tumor originating from adipose tissue. It may be found almost anywhere in the oral cavity where there is adipose tissue, and in the tongue is most often located on the lateral borders. In this area it may eventually interfere with speech and swallowing because it often attains a large size despite its slow rate of growth. The lipofibroma is usually pedunculated but may be found in the submucosa, where it produces a swelling; this latter is the situation in the majority of tongue lesions. The tumor is encapsulated, round or oval, soft, freely movable, and yellow in color with the subepithelial capillaries showing through the thinned mucosa (Fig. 4).

Treatment and Histopathology. Complete surgical removal is indicated. Recurrence is uncommon. Histologically, delicate thin strands of fibrous connective tissue containing capillaries separate the lobules of adult fat cells. In a tumor from the tongue, muscle fibers may be seen at the base. True lipomas are rare and are compsed of a "center of growth where the cells exhibit various stages of fat accumulation."

RHABDOMYOMA

Etiology and Clinical Features. Rhabdomyomas are true tumors arising from the mesenchymal cells which are destined to form striated muscle. They are rare, potentially malignant, and intraorally are most commonly found on the tongue. Young persons, especially children, are most frequently affected. Clinically, rhabdomyomas are firm, circumscribed, elevated nodules lighter in color than the surrounding mucosa (Fig. 5). They are slow growing but may attain a large size. Pain is not a prominent feature.

Treatment and Histopathology. The treatment is similar to that of the leiomyoma, a biopsy again being required to establish the degree of benignity in order to determine the amount of surgery necessary for its removal. In all instances, however, removal must be complete to prevent recurrence. Histologically a marked degree of pleomorphism is noted. The spindle-shaped cells are large and multinucleated. Cross striations in the cytoplasm aid in making a diagnosis. They are not, however, a constant finding.

Fig. 5.   Fig. 6.

Fig. 7.   Fig. 8.

Fig. 5. Rhabdomyoma. Elevated sessile nodule, light in color, and well circumscribed.

Fig. 6. Hemangioma. Nodular elevation on dorsum of tongue, dark red in color. Blanches on pressure.

Fig. 7. Lymphangioma. Well circumscribed, elevated swelling on dorsum of tongue, yellowish brown in color. Lymph fluid can be milked out with pressure.

Fig. 8. Granuloma pyogenicum. Overgrowth of granulation tissue in response to trauma to lateral margin of tongue.

RHABDOMYOSARCOMA

The rhabdomyosarcoma is the malignant counterpart of the rhabdomyoma and is found intraorally in the tongue and palate. It occurs as a growth deep in the tissues and may be of any size. It is not amenable to radiation therapy and therefore must be treated surgically.

PERIPHERAL PLASMACYTOMA

Etiology and Clinical Features. Peripheral plasmacytomas are relatively rare tumors of reticuloendothelial origin and are composed of true plasma cells unassociated with inflammation. They are looked

upon as benign lesions but with reservation, as they are often locally invasive and therefore considered potentially malignant. Plasmacytomas are gray-red in color and are pedunculated or sessile. They are most common in the 30 to 70 age group. The tumor usually occurs singly as a cauliflower-like growth extending above the neighboring mucosa and is seen more often in the floor of the mouth than in the tongue. Tests for Bence-Jones protein are negative. The tumor must be differentiated from the malignant myeloma of bone, which may metastasize to the soft tissues of the oral cavity.

Treatment and Histopathology. Complete surgical removal by a wide clear margin is indicated. Radiation may be used in appropriate cases. Microscopically the tumor is very cellular and is composed of uniform plasma cells with the characteristic cartwheel nucleus. These cells are supported in a reticular stroma of connective tissue. Some mitotic activity may be noted. Cell borders are well defined and debris and reticulum fibers are commonly seen between the cells. The covering stratified squamous epithelium is acanthotic, and keratosis is a common finding.

HEMANGIOMA

Etiology and Clinical Features. The hemangioma is a benign tumor which develops from endothelial rudiments or from the endothelium of blood vessels. It may be congenital or develop as a result of trauma. Hemangiomas are classified as cavernous or capillary, depending upon the size of the vascular space produced, and tend to be larger in the tongue than elsewhere in the oral cavity (Fig. 6). Clinically, they are soft, single or multiple and smooth or nodular. In the tongue they are often globular in appearance and vary in color from red (those near the surface containing numerous large vessels) to purple (those that are deeper in the tissues and contain small capillaries). The lesions nearly always blanch on pressure and pulsation is observed on occasion. Hemangiomas are usually sharply demarcated although sometimes the neighboring tissues are infiltrated. Those in the tongue and the floor of the mouth are subject to trauma which may result in bleeding and ulceration, and they may interfere with and complicate other surgical procedures in the area. A hemangioma may be mistakenly diagnosed as a mucocele or ranula in the floor of the mouth.

Treatment and Histopathology. Surgical removal is frequently indicated in small lesions. Sclerosing solution and radiation are often used in treating larger lesions and may be utilized in combination with surgery or other recognized procedures such as the use of the

endothermy knife. Histologically the excess vascular spaces in both the cavernous and capillary type have a purposeless arrangement and are supported by a fibrous connective tissue stroma. They are lined by active, plump endothelial cells, but not in excess of the number needed.

HEMANGIOENDOTHELIOMA

Etiology and Clinical Features. This vascular lesion also originates from the endothelium of blood vessels and is characterized by an overgrowth of endothelial cells into the lumen or into the supporting connective tissue stroma. It is considered to be potentially malignant, as it does metastasize on occasion and is locally destructive. It is not easily recognized clinically although it resembles other vascular lesions. The hemangioendothelioma may be level with the surrounding structure or elevated, varies in size, is round or oval, firm, and may be the blue-red color of the hemangioma. Pain is seldom produced.

Treatment and Histopathology. Surgery is the treatment of choice and complete excision is indicated. Histologically the tumor is composed of an overgrowth of endothelial cells in the form of sheets and cords into the connective tissue stroma or into the lumen of the vessels. Vascular spaces may or may not be prominent. Mitotic figures and a minimum of cell atypism are observed.

LYMPHANGIOMA

Etiology and Clinical Features. These tumors are composed of endothelium derived from the cells that line the lymphatics. They occur principally in the tongue and are thought by some to be congenital in origin, although many form later in life. The lesions found on the dorsum of the tongue, a common site, are usually unilateral and are often associated with marked keratosis (Fig. 7). This latter produces a rough, white or discolored area. Lymphangiomas may be small and circumscribed or large with a nodular surface. They are usually yellowish brown in color although the presence of vascular channels may produce a bluish tinge. An invaluable diagnostic aid is the ability to force the lymph fluid into the deeper tissue by applying pressure to the lesion. This is known as "milking" the lesion. Ulceration, pain and intermittent drainage are additional symptoms that occur after repeated irritation or trauma to the tumor.

Treatment and Histopathology. Treatment is similar to that of the hemangioma. Microscopically a lymphangioma is composed of distended, connecting, irregular endothelium-lined spaces that are empty or are filled with varying amounts of lymph and, on occasion, a small

number of red blood cells. They are found high in the submucosa and appear to be covered by only a few cell layers of epithelium. Occasionally an unusually large number of vascular spaces are seen in association with the lymphatics.

GRANULOMA PYOGENICUM

Etiology and Clinical Features. Although this lesion is not a true tumor it is included here because it is often misinterpreted by the clinician and pathologist. It appears suddenly as a localized overgrowth of granulation tissue in response to minor trauma (Fig. 8). The granuloma pyogenicum is found frequently in the tongue but seldom in the floor of the mouth, and is a small, elevated, well defined lesion that grows rapidly to full size and remains so indefinitely. It may be pedunculated or sessile, varies in color from red to purple and exhibits a tendency toward hemorrhage. On occasion it appears brown as a result of blood pigment in the tissue due to repeated hemorrhage. Early lesions are usually soft and smooth unless ulcerated; then they are crusted and warty. Older lesions are firm.

Treatment and Histopathology. Complete surgical removal is indicated. Recurrence is frequent and rapid when removal is incomplete. The lesion is composed of granulation tissue with emphasis on endothelial proliferation and the formation of innumerable vascular spaces. The covering stratified squamous epithelium is thin and is frequently ulcerated, with resultant infiltration by inflammatory elements.

SQUAMOUS CELL CARCINOMA

Etiology and Clinical Features. The etiology of squamous cell carcinoma is complex and not well understood. It is generally accepted that there is both an unknown primary factor and known secondary factors or triggering mechanisms. Of the latter, trauma in any of its many forms, syphilis, tobacco, leukoplakia, carbon compounds and azo dyes are most often recognized. Trauma as it relates to the oral cavity may be in the form of ill-fitting dentures, jagged teeth, sharp edges of restorations, clasps that impinge upon soft tissue or any other appliances or objects which cause constant or repeated irritation to an area. Syphilis is also looked upon as a possible secondary factor since it is found much more frequently in patients with carcinoma than those without. It is believed that the resultant ulceration and lowered resistance may be a factor and not the organism per se. Although the use of tobacco has not been proven unequivocally to be a triggering mechanism it is high on the list of suspects, as various substances found in it are known to produce cancer. Leukoplakia,

which occurs as an intact or ulcerated white patch, is considered by many as a stage preceding malignancy. The diagnosis of this lesion must be made on the basis of the histopathology, of which dyskeratosis is the chief diagnostic feature (Fig. 9). Although the term "leukoplakia" has been employed in the past to refer to all white patches in the oral cavity, it is better used in this more restricted sense.

Carcinoma in the tongue may appear as a swelling, a white patch or an ulcerating lesion, with a raised, rounded and firm border (Figs. 10 and 11). Palpation reveals marked induration. These tumors vary from a few millimeters to as much as 8 cm. in diameter, those in the tongue being larger than those in the floor of the mouth. Over half of all intraoral cancers are found in the tongue and of these a majority are found in the posterior two-thirds and at the base. Tumors of this type have not been reported as primary on the dorsum of the middle one-third. The age of the patient may vary from the young to the very old with the mean being approximately 55. Eighty-five to 90 per cent of the tumors are found in the male. Metastases to the regional lymph nodes and distant organs occur in a large percentage of the cases and over half of the patients with cancer of the tongue have metastases to the cervical lymph nodes at time of treatment. Nearly one-fourth of the patients have distant metastases at time of treatment. Only 34 per cent of patients can expect to live 2 years or longer, and 21 per cent for 5 years or longer.

Treatment and Histopathology. These tumors are best treated by complete surgical excision if feasible. They also respond to radiation and on occasion a combination of surgery and radiation therapy is advisable. The highest cure rate, however, is obtained by surgery. All suspected lesions must be biopsied to determine the degree of malignancy and the type of tumor.

Histologically the lesion is composed of malignant squamous epithelial cells which invade the underlying fibrous connective tissue in the form of nests, cords, and strands (Fig. 12). This invasion is often preceded by chronic inflammation. Hyperchromatism, bizarre mitotic figures, and variation in the size and shape of the cells and their nuclei are common. An occasional epithelial pearl is observed.

ADENOCARCINOMA

Etiology and Clinical Features. This malignant tumor arises from the epithelium of the inferior or superior labial glands of the tongue and may also be found in the floor of the mouth on rare occasions. It is a sessile, beefy red, lobulated, firm lesion and grows in folds when located on the tongue (Fig. 13). Pain is experienced as ulceration
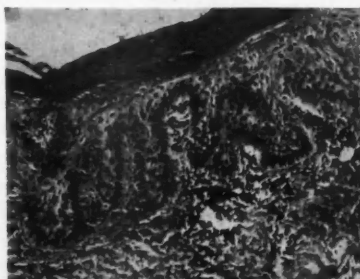
Fig. 9. Fig. 10.
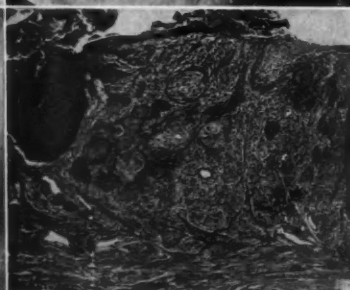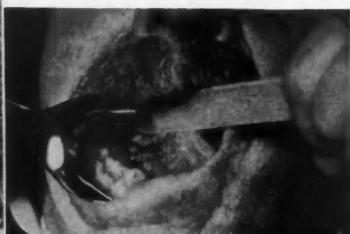
Fig. 13. Fig. 14.

Fig. 9. Leukoplakia. Dyskeratotic changes are noted in the clubbed rete pegs in the form of hyperchromatism, increase in number of mitotic figures and irregularity of the size and shape of the cells and their nuclei.

Fig. 10. Carcinoma of the tongue. The border of the lesion is raised, rolled, and indurated while the center is ulcerated and bleeding. Lesion was painful.

Fig. 11. (center, left). Carcinoma of the floor of the mouth. An ulcer with slightly raised, rolled edges. The central part of the lesion is yellow and necrotic in appearance, with numerous small ulcerated areas.

Fig. 12. (center, right). Squamous cell carcinoma. Invasion of the submucosa in the form of nests of malignant epithelial cells is noted.

Fig. 13. Adenocarcinoma of the tongue. Sessile, lobulated lesion, firm and red in color.

Fig. 14. Mixed tumor. Large ulcerated lesion of the palate which is firm and interferes with mastication and speech.

occurs but in general, subjective symptoms are lacking. Because the lesion does not metastasize early the prognosis of this type of malignancy is considered to be relatively good.

Treatment and Histopathology. Treatment consists of complete surgical excision with a wide border of normal tissue. On occasion radiation therapy or a combination of radiation therapy and surgery may be employed. Microscopically the epithelial cells are small and basal in character, but may vary in size and shape. They tend to form acinar structures. The tumor is not encapsulated.

MIXED TUMORS

Etiology and Clinical Features. Mixed tumors are true neoplasms of epithelial origin and are thought to arise from adult glandular epithelium. Approximately 10 per cent of these tumors are found in the floor of the mouth (9 per cent in the submaxillary glands, 1 per cent in the sublingual salivary glands), and a few in the tongue. They are most prevalent in middle age and over half are seen in females. Both benign and malignant varieties exist, with the former most common. Numerous recurrences are characteristic of the lesion. Clinically these tumors, when in superficial locations, are round, smooth and freely movable. When deeply placed they are indistinct in outline. Mixed tumors are of intermittent or slow growth and may be present for long periods of time. There are reports of lesions of this type being present for as long as 20 years before treatment. They may cause local discomfort due to increased pressure in the area and although ulceration is not the rule it sometimes occurs (Fig. 14).

Treatment and Histopathology. These lesions are best treated by complete surgical removal. They tend to recur, owing chiefly to finger-like growths of the tumor beyond the apparent capsule. These are frequently cut off and left behind at time of surgery. A lesion suspected of being a mixed tumor should be biopsied in order to confirm the diagnosis; however, because of its marked ability to recur it would seem advisable to perform the biopsy (frozen section) at time of removal of the entire tumor. Histologically there is great variation in these tumors. Cellular elements may predominate and bone, cartilage or mucus can be variants. In the malignant mixed tumors the epithelium is the malignant element.

BASALOID MIXED TUMOR (ADENOCYSTIC BASAL CELL CARCINOMA)

Etiology and Clinical Features. Basaloid mixed tumors are malignant and originate from glandular epithelium. They are as frequent in their occurrence in the floor of the mouth as they are in the parotid.

Clinically these tumors simulate the benign mixed tumor. There are, however, certain subjective and objective symptoms that may aid in making a differential diagnosis. The basaloid mixed tumors often produce pain early in the course of their growth. Facial nerve palsy, cervical lymph node metastases and fixation to the mandible are not uncommon. Although adenocystic basal cell carcinomas are of slow growth there is only a 20 per cent 5 year cure rate at the present time.

Treatment and Histopathology. Surgery is the treatment of choice. It is believed that a change from the present practice of conservative surgery to radical surgery will produce better cure rates. Radiation therapy is generally unsatisfactory. The chief histologic feature is the arrangement of rather small, darkly staining cells in anastomosing cords between which are rounded acellular areas that may be empty or may contain mucus or other material.

MUCOEPIDERMOID MIXED TUMORS

Etiology and Clinical Features. All mucoepidermoid mixed tumors are malignant lesions arising from the ductal epithelium of salivary glands and they may be either low grade or high grade malignancy. Although this is chiefly determined by the histopathology, several clinical features may be of help. The tumors of low grade malignancy occur as a painless swelling. Those that are of high grade malignancy commonly produce pain as an initial symptom, and pain may, as a matter of fact, antedate discovery of the lesion by several months. The clinical course of this latter tumor reflects a much more vigorous rate of growth. Regional lymph node metastases may or may not be present. All these lesions occur at an earlier age than the mixed tumor. Most are found in the parotid but about 10 per cent are seen in the floor of the mouth (submaxillary). None have been observed in the sublingual area. They are smaller in size than the mixed tumors. Clinically they are ovoid in shape, firm, fairly well circumscribed and not infrequently fixed to the underlying tissues.

Treatment and Histopathology. Complete surgical excision with a wide margin is indicated. Radiation therapy is of little value. The prognosis of the low grade tumor is good, whereas that for the high grade tumor is rather poor. Microscopically, three types of cells are observed: the epidermoid cell, the mucous cell and an "intermediate" cell, which resembles certain cells of the salivary gland duct.

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Tumors of the Buccal and Labial Mucosa

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It is difficult to ascertain the frequency of occurrence of various types of tumors of the oral mucosa because of the problems encountered in limiting the definition of these growths (including or excluding the non-neoplastic lesions such as papillary fibrous growths, mucoceles and leukoplakia) and because of the paucity of accurate data on their incidence in unselected groups. Surveys from large cancer hospitals, from the Armed Forces Institute of Pathology or from consulted clinics would be biased. At best, we can gain general impressions from surveys in various institutions and bear in mind the many defects inherent in such methods of analysis.[3,6,8,12]

ORIGIN OF TUMORS OF THE BUCCAL AND LABIAL MUCOSA

Nearly all tumors found in buccal and labial mucosa arise as primary new growths from normal mucosal structures.

The tissue that covers the labial and buccal mucosa is a non-keratinized, stratified squamous epithelium. Immediately beneath the epithelium is a relatively dense layer of fibrous connective tissue. Deeper there are dense bundles of collagenic tissue extending to the buccal and labial musculature. Small groups of mixed salivary glands are situated in the submucosa, their excretory ducts rising to the surface. Of special interest is Stensen's duct. Small groups of fat cells, blood and lymph capillaries, and nerves are abundant. Sebaceous glands are observed, at least to some degree, in the buccal mucosa of over 80 per cent of adults. Their presence has been called Fordyce's disease although their presence now is considered normal rather than pathologic.[4] Heterotopic lymphoid tissue may be observed occasionally as a few lymphoid follicles or even complete lymph nodes.[5,9] Tumors can develop in any of these tissues. Benign tumors composed

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largely of surface epithelium (squamous cell papillomas), fibrous connective tissue (fibromas), fat (lipomas), blood and lymph vessels (angiomas), nervous tissue (neuromas, neurilemmomas and neurofibromas), muscle tissue (myomas) and all of their malignant counterparts may be observed. Neoplasms arising from salivary glands are not too unusual in this area and if we are to include mucus retention cysts, they comprise a considerable proportion of labial lesions.

PAPILLOMATOUS LESIONS—TRUE AND FALSE

Within this wide category are included squamous cell papilloma, fibroepithelial lesion or irritation fibroma, epulis fissuratum or denture injury enlargement, and true fibroma.

Squamous cell papilloma is a tree-like or arborescent growth that usually has a narrow stalk-like base. It consists principally of stratified squamous epithelium covering a thin core of well vascularized connective tissue. It is always benign and does not recur when removed either by surgery or electrocautery. It has been cited by some as being a premalignant growth but this author has never seen an authenticated case. It is far more likely that there is confusion of this quite benign lesion with the insidious papillary, verrucous, or exophytic squamous cell carcinoma. Squamous cell papilloma occurs far more commonly on the buccal mucosa, uvula, palate, and tonsillar areas than on the mucous membrane of the lip. It is usually single but may be multiple, especially in the palatal region. It is more common in older persons, although the author has seen one involving the tonsillar pillar in a newborn child.

The fibroepithelial lesion, or irritation fibroma, is a pseudo-neoplastic mass, usually broad based or sessile. It is the most common lesion arising from the buccal and labial mucosa. The lesion can range in size from a few millimeters to almost 2 cm. in diameter. Characteristically it is soft, rounded, and covered by normal appearing mucous membrane (Fig. 1). There appears to be a definite relationship of this tumor to trauma. Commonly it is observed opposite a naturally occurring diastema or one produced by tooth extraction. It may be generated by heaping up of normal mucosa due to the unconscious habit of applying negative pressure to this interdental space. Direct tooth trauma has been implicated but whether this has a cause or effect relationship has not been ascertained.

Microscopically the lesion appears as a hypertrophic or hyperplastic mass of normal mucosa rising above the normal cheek or lip surface. Its microscopic architecture cannot be differentiated from normal mucosa or true fibroma. Where biting trauma is a factor,

Fig. 1.   Fig. 2.

Fig. 3.   Fig. 4.

Fig. 1. Traumatic, or irritation, fibroma, by far the most common benign "neoplasm." It frequently occurs on the buccal or labial mucosa opposite a diastema.

Fig. 2. Denture trauma enlargement, which also results, like the irritation fibroma, from chronic irritation. Fibroblastic proliferation results in tumor-like growths that involve the labial or buccal mucosa.

Fig. 3. Hemangioma, a new growth composed of blood vessels. It may be minute or so large as to include half of the maxilla. It has a characteristic red or purple color.

Fig. 4. Pyogenic granuloma, an angry red growth that can appear anywhere on the oral mucosa or skin. It represents the excessive proliferation of granulation tissue in response to minor physical trauma or hormonal influences. It is indistinguishable from pregnancy tumors or infected capillary hemangiomas.

surface keratinization, inflammation, and even frank ulceration are common.

Epulis fissuratum, or denture injury enlargement, is a common lesion in patients who wear or have worn dentures which no longer fit. It is really a lesion of the gingiva and mucobuccal fold but may upon occasion be extremely redundant and extend to the buccal mucosa proper (Fig. 2). Vascularity is variable, depending upon the chronicity of the lesion. Some inflammation is nearly always present.

True fibroma is far less common than is cited in routine surveys. It is extremely difficult to differentiate the true neoplasm from the fibroepithelial lesion, and only when the tumor is extremely cellular or consists of randomly arranged collagen bundles and cells can we suspect the diagnosis. It is not unusual for other tissues to be alloyed, producing the lipofibroma, or osteofibroma. Myxomatous degeneration sometimes occurs and may be incorrectly diagnosed as myxoma.

VASCULAR NEOPLASMS—TRUE AND PSEUDO

Separation of true angiomas from other non-neoplastic angiomatous proliferations is not at all clear cut. True angiomas arise out of neoplastic proliferation of their own vessels. These tumors, when arising from a mucosal surface, may be present at birth or more frequently develop in later life.

The hemangioma is composed of large numbers of vascular blood-filled channels (Fig. 3). These may be minute (capillary), intermediate (venous), or of large size (cavernous). Seldom, however, is a tumor composed of only a single type. Frequently they are encountered on the lips, buccal mucosa, and tongue. They may be only a few millimeters in diameter or involve almost half of the face, following the trigeminal nerve distribution (Sturge-Weber syndrome) and involving the lips, cheek, palate, etc.[10] The color depends on the type of vessels involved and their distance from the surface. Small lesions may be removed by excisional biopsy but larger ones are best treated by injection of sclerosing solutions or by irradiation. These tumors must also be differentiated from hematomas which result from acute trauma, and from juvenile hemangiomas or benign hemangioendotheliomas that are sometimes seen in infants. Because of their location, all of these lesions are subjected to physical trauma and may evince some degree of inflammation.

A lymphangioma occasionally arises in the buccal or labial mucosa. The lymphangioma is most frequently present at birth. It is composed of numerous quite superficial lymph-filled vessels. It is usually more extensive than the hemangioma and surgical removal is not always desirable. Sclerosing solutions are used whenever possible.

The pyogenic granuloma, or "capillary hemangioma-granuloma type," is composed of an exuberant mass of granulation tissue (Fig. 4). It apparently arises in response to trauma which in many instances is quite minor and not at all proportional to the size of the lesion. In our experience it is most commonly seen in persons below 21 years of age, but Kerr, in his large series, did not observe this age relationship.[7] The tumor-like mass may be only a few millimeters in diameter or

may assume large proportions. The patient is usually unaware of it in its initial stages. It is usually first noticed as a painless, easily traumatized, bleeding mass. It rapidly becomes ulcerated and secondarily infected. It is composed of huge numbers of proliferating blood capillaries intermixed with some fibroblasts and inflammatory cells. Treatment consists of simple, though rather deep, surgical excision. Incomplete removal often results in regrowth.

CYSTS AND TRUE TUMORS OF MINOR SALIVARY GLANDS

Beneath the epithelium of the mucous membrane of the lip and cheek there are numerous groups of salivary glands. They are mixed glands, but most of them are of the mucous type. Sometimes they give rise to cysts (mucoceles or mucus retention cysts) or true neoplasms.

The mucocele is second in importance only to the fibroepithelial papilloma among benign lip and cheek lesions. It is not a true neoplasm but because of its glistening bluish color it may be confused with an angioma. The cyst may arise following severance of the excretory duct by physical trauma. It is rather common subsequent to placement of orthodontic bands or minor lip surgery. The fluid which is elaborated by the gland cannot be released and a cystic swelling results. The cyst is subject to trauma and may rupture. Usually they are removed in toto by surgery or electrodesiccation.

True tumors of minor salivary glands can also arise. There appears to be some selectivity of site. Roughly, the ratio of lesions of the palate, lip, and buccal mucosa is 10:5:1.[2]

The tumors of the minor glands, like those of the major salivary glands, may be benign or malignant. The most common types are *mixed tumor, mucoepidermoid tumor, cylindroma* or adenoid cystic basal cell carcinoma, and *papillary cystadenoma.* The mixed tumor clinically may be benign or malignant. In the minor salivary glands it is not well encapsulated, and unless widely excised it is prone to recur. The mucoepidermoid variety should always be considered malignant.[11] Generally the clinical course follows that which would be predicted from the histologic appearance, but sometimes the microscopically benign lesion will metastasize quite widely. Herein lies the reason for assuming that even the most harmless-appearing variety may have malignant potentialities. The cylindroma or adenoid cystic basal cell carcinoma is more than just locally destructive. Papillary cystadenoma is a quite benign tumor and may be removed conservatively. All other salivary gland neoplasms should be widely excised. It is well to remember that minor salivary glands are not encapsulated so no natural delineation exists.

SQUAMOUS CELL CARCINOMA AND "PRECANCEROUS LESIONS"

Squamous cell epithelioma occurs on the lower lip more often than on all other areas within the mouth, but the labial mucosa is not the primary site of involvement. The usual point of origin is the vermillion area of the lower lip to one side of the midline. This lesion may expand slowly—as in the case of verrucous or exophytic squa-

Fig. 5.   Fig. 6.

Fig. 5. Epidermoid carcinoma of labial mucosa. The patient stated that he had a "cold sore that didn't heal up for three weeks." From the size of the lesion, the duration as stated by the patient is highly doubtful.

Fig. 6. Epidermoid carcinoma of buccal mucosa. Patient had excessively poor oral hygiene, numerous broken-down teeth. The tumor extended back to the ramus of the jaw before the patient sought help.

Fig. 7. Epidermoid carcinoma arising in leukoplakia. Whenever a patch of leukoplakia cracks or ulcerates it should be immediately biopsied.

Fig. 7.

mous cell carcinoma—or may rapidly infiltrate and extend to the mucosal surface—as in the case of the more common ulcerative or endophytic squamous cell carcinoma (Fig. 5). The involvement occurs, therefore, when the disease becomes advanced, the lesions larger, and the prognosis far less favorable. Although rarely arising at this site, nearly all labial mucosal malignancies are nevertheless squamous cell carcinomas, but there is seldom early metastasis. It has been estimated that the buccal mucosa is the site of about 9 per cent of all intraoral squamous cell carcinoma.[1] The most frequently involved

area is the middle third of the cheek, along the line of occlusion of the second premolar and first and second molar teeth, just below the orifice of Stensen's parotid duct (Fig. 6). It is not unusual for this papilla to be involved by extension of the cancer. As we proceed from the lips to the tonsillar pillars the prognosis becomes progressively poorer, as the cancer rapidly invades the deep structures of the neck and metastasizes more rapidly owing to a richer lymphatic drainage. Carcinoma of the buccal mucosa does, however, have a better prognosis than carcinoma of the floor of the mouth.

Leukoplakia is rather commonly observed in this area. In the past, the term "leukoplakia" was used to mean any white patch on a mucosal surface. Literally this is what the word means, but not all white patches are identical in nature. While simple hyperkeratosis, thrush, syphilitic mucous patches, psoriasis, lichen planus, dyskeratotic mucosal lesions, etc. all clinically are white patches, they are hardly similar processes. There have been numerous attempts to clarify this situation by using specific microscopic characteristics to distinguish the two most clinically confusing lesions, simple hyperkeratosis, or pachyderma oralis, and true leukoplakia (Fig. 7). It is believed that only the latter may give rise to cancer. They may be easily, and only, differentiated under the microscope. Biopsy must be performed, for clinical impression is not enough. There is no good evidence that simple hyperkeratosis ever changes to leukoplakia or gives rise to any true neoplasm.

The term "precancerous" is used to designate a lesion or condition which, under influence of certain unknown causal factors, will transform into frank carcinoma. This does not mean that all cases of true leukoplakia will eventuate in cancer. Neither is the converse true, that all squamous cancer arises in areas of leukoplakia. Since one cannot tell clinically which white patch is going to become malignant, the question of prognosis must be presented to the pathologist. If dyskeratosis or irregular maturation of cells is present, malignancy may follow.

MISCELLANEOUS TUMORS

A wide variety of miscellaneous tumors arises in the labial and buccal mucosa but they occur so seldom that they should merit little attention in this brief survey.

Lipomas arising from small groups of submucosal fat cells or from the buccal fat pad are occasionally observed. Nerve tumors, such as *neuromas, neurofibromas,* and *neurilemmomas* may arise from the many nerve fibers that course through the connective tissue. Lymphatic tissue may become hyperplastic and become clinically manifest

as *heterotopic nodes* or may become the site of a *lymphoma*. Very rarely, a *melanoma* arises in the buccal or labial mucosa. This tumor, wherever it arises, is always highly malignant.

SUMMARY

By far the most common "tumor" arising in the labial and buccal mucosa is the fibroepithelial lesion or irritation fibroma. The next most common is the mucocele. Neither is a true neoplasm. The most common oral malignancy affecting this area is squamous cell carcinoma. It seldom arises as a primary lesion of the labial mucosa but has its origin on the vermillion area and extends to the mucosal surface. Buccal mucosal carcinoma metastasizes more widely than carcinoma of the lower lip and its prognosis becomes poorer as it extends toward the oral pharynx.

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Tumors of the Palate

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Primary tumors of the hard and soft palates arise from the surface epithelium, from the mucous glands and connective tissue of both palates, from pigment forming cells of neural crest origin, from vascular tissue, and from the bone of the hard palate. Secondary tumors of these structures usually occur as direct extensions from tumors of adjacent structures such as the maxillary sinus and the nasopharynx. Secondary involvement of the palate from more distant sites via blood stream metastases is extremely rare. Tumors of the palatal gingiva will not be considered because they are essentially similar to the lesions of the other gingiva.

TUMORS OF SURFACE EPITHELIUM

The surface epithelium of the hard palate and the oral side of the soft palate is of the stratified squamous type, while that of the pharyngeal side of the soft palate is ciliated, pseudostratified columnar. The latter is characteristic of respiratory epithelium. A transitional zone between the two types exists as the epithelium is reflected from the oral to the pharyngeal side. This zone could be the point of origin of some so-called transitional cell carcinomas of the soft palate.

The surface epithelium of the hard and soft palates gives rise to benign and malignant tumors. The former are squamous cell papillomas and the latter are squamous cell or transitional cell carcinomas.

Squamous Cell Papilloma

The squamous papilloma appears to occur more frequently in the hard palate than in the soft palate because inflammatory papillary hyperplasias associated with relief chambers of artificial dentures

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are often included in this group. These lesions occur in the midline of the hard palate. They are elevated, reddish, lobulated or papillary lesions, which are attached by a broad base. The true neoplastic papilloma, on the other hand, may be seen anywhere on either palate. It is frequently whitish in color, attached by a narrow or broad base, and an obvious local irritant is lacking. It may appear flattened if it occurs under a denture. Management of inflammatory papillary hyperplasias includes elimination of the irritating denture, a short period of observation of regression of the lesion, and finally excision of any portion of the lesion which fails to resolve. Lesions which are interpreted clinically as true papillomas should be excised in toto, including the base. All excised tissue should be placed immediately in a suitable fixative (e.g., 10 per cent Formalin) and submitted to a pathology laboratory for histopathologic study, not only to confirm or refute the clinical diagnosis but also to determine whether or not the lesion has been completely removed.

The relationship of these two lesions to their malignant counterpart, squamous cell carcinoma, is not completely clear. Hobaek[3] reported four cases in which squamous cell carcinoma developed under the suction chambers of artificial dentures. Sharp, Bullock, and Hazlet,[5] who reviewed 51 cases of carcinoma of the palate, stated that "The mechanical trauma of negative pressure created by excessive relief of the palate of a denture . . . has a remarkably low relationship to carcinoma." Thoma[6] did not separate inflammatory and neoplastic papillomas in estimating prognosis, and appears to be of the opinion that both lesions are potentially malignant. He apparently feels that some slow-growing papillary squamous cell carcinomas are misinterpreted as papillomas, as opposed to Ewing, who stated that a "gradual transformation of a benign papilloma into carcinoma has been fully demonstrated." In either case, it appears safe to assume that lesions which appear clinically as papillomas must be considered as potentially malignant and managed accordingly.

Squamous Cell Carcinoma

Squamous cell carcinoma of the hard and soft palates arises as a result of neoplastic proliferation of the stratified squamous epithelium which covers these structures. As in the remainder of the oral cavity, this is the malignant tumor that the dentist is most likely to encounter. It accounts for about 10 per cent of all intraoral carcinomas, according to Sharp, Bullock and Hazlet[5] and Bernier.[1]

As in other malignancies, the etiology of this lesion is not precisely known. Although Sharp, Bullock and Hazlet found a very low cor-

relation between relief chambers of dentures and carcinoma of the palate, they did find a rather significant correlation between carcinoma of the junction of the hard and soft palates and an irritating posterior denture border. Further study is necessary to determine the precise cause and effect relationship here, for it could well be that the irritation by the denture occurred after the malignancy was established. Other extrinsic factors which have been considered in the etiology of palatal squamous cell carcinoma are pipe smoking, cigar
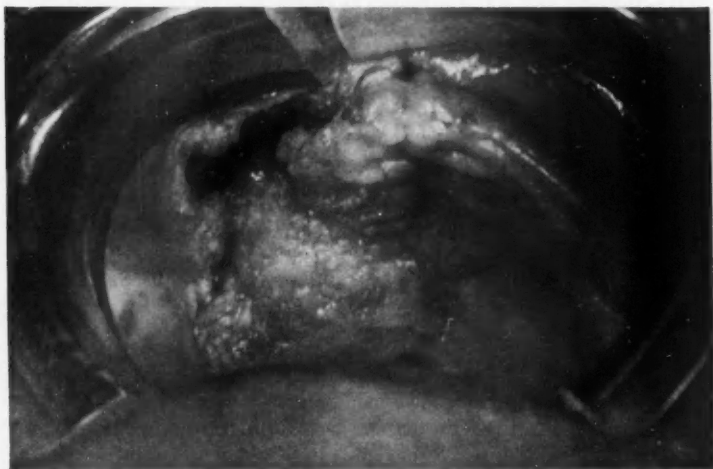
Fig. 1. Neglected squamous cell carcinoma of the hard palate which has destroyed the entire alveolar ridge on one side and invaded the maxillary sinus.

smoking, and the habitual use of scalding liquid foods. The available evidence indicates that different people respond differently to these irritants. In some cases, there appears to be little or no observable effect; in others, keratotic lesions such as leukoplakia and stomatitis nicotina develop; in still others, frank carcinoma becomes evident and appears to have a direct relationship to the irritant. These variable reactions may be the result of the time factor or of intrinsic susceptibility to extrinsic irritants. Available evidence indicates that nutritional deficiencies and hormonal imbalance, which are frequently associated with the ageing process, may play a significant role.

The clinical appearance of palatal squamous cell carcinoma varies considerably. Advanced infiltrating types often exhibit the classic textbook appearance of a malignant tumor. These lesions are non-elevated and circular, with necrotic centers and rolled, raised, indurated bord-

ers. However, even with this appearance a final diagnosis cannot be made by clinical appearance alone because the infective granulomas may exhibit a similar appearance. Other carcinomas appear as irregular granular (Fig. 1) or papillary masses. The latter may show considerable outgrowth and thus simulate the clinical appearance of the broad-based papilloma. Both types invade the underlying connective tissue and metastasize to the submaxillary and cervical lymph nodes. In the hard palate, the bone is readily invaded and penetrated so that invasion of the maxillary sinus and nasal chamber is common.

The management of this lesion is based first on the establishment of the diagnosis with the aid of biopsy. As soon as the diagnosis has been confirmed, radical surgery by a qualified cancer surgeon is the treatment of choice. Some poorly differentiated lesions respond to radiation therapy. When the tumor has been eliminated by surgery, prosthetic reconstruction should be considered by the dentist, since the maxilla is a most favorable site for this procedure. Some opportunity for the latter procedure exists because 5 year cure rates vary between 31 and 57 per cent,[4] depending upon whether the diagnosis and treatment occur early or late. Unfortunately, the other 43 to 69 per cent were dead or exhibited recurrences within 5 years.

Transitional Cell Carcinoma

Transitional cell carcinoma is a malignant tumor which arises from the surface epithelium of the palate, but exhibits little or no tendency to become differentiated. Some of these lesions may arise in the transitional zone at the reflection of the soft palate. While the origin of some transitional cell carcinomas can be explained on this basis, others apparently arise on the oral or pharyngeal side of the soft palate at some distance from the transitional zone or even occasionally on the hard palate. These lesions must be explained on the basis of metaplasia or extreme dedifferentiation. The former applies to the respiratory epithelium of the pharyngeal side of the soft palate, the latter to the stratified squamous epithelium of the oral side of both palates. These lesions frequently occur in close proximity to the lymphoid tissue of Waldeyer's ring. When significant infiltration of the stroma by lymphocytes is evident in histologic sections, the term lymphoepithelioma becomes appropriate. Hence, one can consider the latter as a variant of transitional cell carcinoma. The clinical appearance of these lesions is similar to squamous cell carcinomas and they can be differentiated only by microscopic study. This distinction is significant because the transitional types are more amenable to radiation therapy.

TUMORS OF MUCOUS OR MINOR SALIVARY GLAND ORIGIN

A large group of tumors arises from the mucous glands located in the submucosa of the posterior portion of the hard palate and in the soft palate. These lesions include the relatively common benign mixed tumors and their malignant variants, as well as the very rare adenoma and its malignant counterpart, adenocarcinoma. Separation of these lesions on the basis of their clinical appearance is not possible, regardless of one's diagnostic acumen. They appear clinically as superficial or more deeply situated nodules in the hard or soft palate, covered by a relatively normal appearing mucosa. The superficial
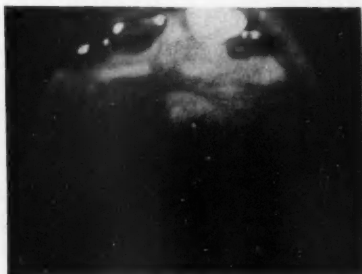
Fig. 2.   Fig. 3.

Fig. 2. Benign mixed tumor of the salivary gland type, located on the lateral aspect of the hard palate. Note the normal-appearing covering mucosa.

Fig. 3. Bulky tumor involving hard and soft palates and posterior alveolar ridge. Note keratotic areas and small ulcerations of the covering epithelium. Biopsy diagnosis was benign mixed tumor, indicating that surface changes were caused by secondary trauma. Nodule in buccal mucosa was a fibroma.

types may be attached by a narrow pedicle (Fig. 2). Ulceration of the surface epithelium (Fig. 3) is indicative of secondary physical trauma or malignancy. Fixation of the lesion to the overlying mucosa or underlying bone or soft tissue is also indicative of malignancy. Occasionally, an adenocarcinoma will appear as a non-elevated ulcerous lesion with indurated borders.

Mixed Tumors

The second most frequent site of occurrence of mixed tumors of the salivary gland type is the hard and soft palates. The vast majority of these lesions are quite benign and grow very slowly. In a series of several hundred cases reviewed by Cheyne, Tiecke and Horne,[2] the average duration at the time of diagnosis was almost 11 years. The shortest duration was 5 months and the longest 41 years. In spite of

their apparent benignancy, most of them show a high tendency to recur following excision and a small number exhibit malignant clinical behavior. Some of the malignant types show the time-honored histopathologic criteria for malignancy so that their clinical behavior can be accurately predicted by the pathologist. However, others have shown, in the past, a poor correlation between their microscopic appearance and their clinical behavior. This problem resulted in the recognition of such new entities as the basaloid mixed tumor and the mucoepidermoid tumor. While these lesions often do not exhibit the usual microscopic criteria for malignancy, the skilled pathologist now recognizes them for what they are and is able to predict their clinical course with increasing accuracy.

The difference in clinical behavior of benign mixed tumors and their malignant variants is quite striking. The former will remain localized for long periods of time even if untreated. Invasion of the surrounding tissues does not occur, although slight penetration of the capsule may be noted in histologic sections. Metastases never occur. In the malignant types invasion of the capsule occurs, followed by invasion of the surrounding tissues, or metastasis to the regional lymph nodes and the lungs. The question of whether benign mixed tumors change into malignant mixed tumors is unsettled. In this author's opinion most of the malignant forms are malignant from the beginning, but may be misdiagnosed as benign. Occasionally, a tumor of long duration and slow expansive growth suddenly changes its character and breaks through its restraints, resulting in invasion of contiguous tissue and metastases. This rare occurrence can be best explained on the basis of malignant change in a previously benign mixed tumor.

The management of mixed tumors of the palate is usually surgical. The diagnosis is established by histopathologic study of either an incisional or an excisional biopsy. The latter is preferable if no deformity will result from the procedure because the treatment is complete if the specimen exhibits a benign character on microscopic examination. If it proves to be a malignant variant, a second more radical operation by a qualified cancer surgeon is indicated. Radiation therapy is sometimes used in long-neglected, inoperable malignancies as well as some recurrent cases.

Adenoma and Adenocarcinoma

Adenomas are benign tumors of glandular tissue. Since there is an abundance of mucous gland tissue in both the hard and the soft palates one would expect to see this lesion in this location. However,

if adenomatous types of mixed tumors are eliminated, one would be hard put to find reports of any significant number of these lesions. The malignant counterpart of this lesion, the adenocarcinoma, apparently is somewhat more common. The latter must be distinguished from the malignant variants of the mixed tumor which show adenomatoid features. The clinical appearance of these lesions is generally similar to that of the mixed tumors, although some adenocarcinomas tend to resemble the clinical appearance of surface squamous cell carcinoma. The distinction is established by microscopic study. It is a significant distinction because the adenocarcinoma is a more indolent lesion. It grows relatively slowly, metastasizes late, and thus is more easily controlled by proper therapy.

CONNECTIVE TISSUE TUMORS

Connective tissue tumors of the palates include the fibroma, neurofibroma, and their malignant counterparts, fibrosarcoma and neurogenic sarcoma.

Fibroma and Fibrosarcoma

Palatal fibromas arise from the deeper layers of the connective tissue of the palatal mucosa and from the periosteum of the palatal bone. A small number are true fibromas, but the vast majority are exuberant scar tissue similar to the keloid of the skin. These lesions appear clinically as spherical or irregular nodules which may be either sessile or pedunculated, and either pink or gray in color. The pink ones are usually soft and the gray ones hard. This difference often corresponds to their microscopic appearance, the former exhibiting increased vascularity and cellularity and the latter minimum cellularity and much collagen. The highly cellular types must be distinguished by histopathologic study from their malignant counterparts, the fibrosarcomas. Local excision is the treatment of choice for fibromas and radical surgery is indicated in the case of the fibrosarcoma. Fibrosarcomas of the oral cavity behave in a more indolent manner than similar lesions elsewhere in the body and hence are more frequently cured.

Neurofibroma and Neurogenic Sarcoma

The neurofibroma arises as a result of neoplastic proliferation of fibroblasts which are present in the sheaths of palatal nerves. It may appear as a single nodule in the palate or as part of the generalized

neurofibromatosis of von Recklinghausen's disease of the skin. Patients with the latter condition may exhibit several hundred nodules in the skin. Often one or more proves to be neurogenic sarcoma microscopically. The palatal lesions are usually benign. A favorite location is in the area of the posterior palatine foramen, where there may be some erosion of the underlying bone. Single benign lesions are managed by local excision. Multiple lesions are difficult to manage.

TUMORS OF PIGMENT-FORMING CELLS

Pigment-forming cells, or melanoblasts, are normally found in the eye, the skin, and the mucous membranes. Therefore they are normal constituents of the palatal mucosa. They arise from neural crest tissue and then migrate peripherally during embryonic development. Tumors of these cells include the benign *dermal nevus,* the potentially malignant *junctional nevus,* and the malignant *melanoma.* All three of these lesions are very rare intraorally. The benign nevi appear clinically as non-elevated or elevated pigmented lesions. The elevated types are more likely to become irritated and these types are more likely to exhibit junctional activity in histologic sections. ("Junctional activity" refers to active melanoblastic proliferation in the area of the junction between the surface epithelium and its supporting connective tissue.) Any pigmented lesion of the palates or elsewhere which has a history of active clinical growth that is not correlated with general body growth should be presumed to be malignant melanoma until proven otherwise. This is so because the benign nevi are actually congenital malformations which grow only when the body grows and stop growing when the body stops growing. Fortunately, the vast majority of pigmented lesions are benign, but if malignant melanoma is suspected on the basis of careful clinical evaluation, immediate consultation with a competent cancer therapist is imperative, for this is one of the most highly malignant tumors.

TUMORS OF VASCULAR TISSUE

The *hemangioma* and *hemangioendothelioma* arise in the palates as a result of proliferation of the vascular endothelial cells. The former is a malformation in which the blood vascular spaces are overemphasized. Most hemangiomas are congenital but apparently some are acquired later in life. A few exhibit an aggressive behavior with a tendency to infiltrate surrounding tissue, but they never metastasize. The hemangioendothelioma, on the other hand, must be considered a true neoplasm of the vascular endothelium because of its generally

aggressive behavior and the fact that metastases have been reported in a few cases. Clinically, the hemangioma is usually an elevated purplish or reddish lesion which is filled with blood. The blood can be forced out of the lesion by digital pressure. Hemangiomas of the soft palate tend to be quite bulky, whereas hard palate lesions tend to be flat. Hemangioendotheliomas may contain little or no blood.

TUMORS OF THE PALATAL BONE

Primary tumorous lesions of the palatal bone include the extremely common *torus palatinus*, the *osteoma*, and *fibrous dysplasia of bone*.
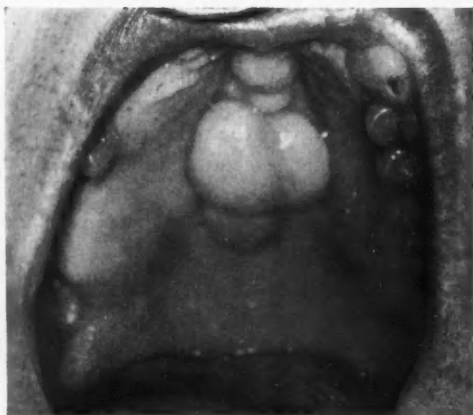
Fig. 4. Large lobulated torus palantinus. In this type, excision is indicated to facilitate denture construction.

Some pathologists consider the latter to be a neoplastic process. All three lesions are benign. Malignant tumors of the palatal bone are extremely rare. The torus, which is not neoplastic, appears clinically as a bony outgrowth in the midline of the hard palate covered by a thin mucosa. It occurs in about 20 per cent of the general population, is more common in women than in men, and may be flat, spindle, nodular, or lobulated in shape (Fig. 4). Mixed tumors of the hard palate are occasionally confused with these lesions by the inexperienced examiner. When they become ulcerated as a result of trauma, they may be the basis of cancerophobia in patients. The torus palatinus is removed by local excision only when it interferes with denture construction. According to Thoma,[6] "the true osteoma is distinguished from the torus by its lateral location and single site of origin." Local excision is the treatment of choice. Fibrous dysplasia is

characterized by proliferation of osteoplastic fibrous tissue in the bone marrow at the expense of the surrounding bone. Expansion and distortion of the bony palate occurs. Diagnosis is based on clinical, roentgenographic, and histopathologic findings. Treatment consists of surgical reduction of the gross deformity.

SECONDARY TUMORS

Sometimes the hard and soft palates are invaded by tumors which arise in the maxillary sinuses or the nasopharynx. These lesions arise from the surface or glandular epithelium of the latter structures and are invariably malignant. They may be *squamous cell carcinoma*, *transitional cell carcinoma*, or *adenocarcinoma*. Sarcoma is extremely rare. The dentist may have the first opportunity to diagnose these lesions because the first clinical sign may be a swelling in the hard palate which the patient thinks is associated with the teeth. Indeed, loose, sore teeth may accompany the palatal swelling. Another clinical sign which brings the patient to the dentist is the failure of a previously well adapted upper denture to seat properly. Obviously, these criteria also apply to primary palatal tumors. Roentgenograms are of great value in helping to establish the diagnosis in secondary tumors because bone destruction has already occurred when the lesion becomes manifest intraorally. However, the definitive diagnosis again depends primarily on microscopic study of a biopsy specimen. Management is in the hands of the cancer surgeon or the radiation therapist or both.

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Tumors of the Gingiva

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The gingival tumors are growth disturbances of both benign and malignant nature. When they occur at birth they are termed congenital. The benign tumors are slow growing, well circumscribed encapsulated lesions composed of excessive normal tissue resembling a parent tissue. For example, a hemangioma is a tumor of excessive tissue resembling normal blood vessels. When such tumors occur at birth they are called congenital vascular nevi. The benign tumors of the gingiva include excessive non-functional growth of stratified squamous epithelium, fibrous tissue, vascular tissue, peripheral nerve, melanoblasts, and periosteum.

Probably the most frequently occurring benign tumors are the reparative granulomas. These lesions are associated with some injury to the gingiva which results in an inflammatory process accompanied by repair. The growth disturbance in this case involves repair tissue. There is excessive, but limited, proliferation of the fibroblasts and capillaries, which gives bulk to the tumor. Equally important is the preponderance of one of such tissues and their product which imparts clinical and histologic character to the tumor.

Malignant tumors of the gingiva are principally the squamous cell carcinoma and rarely the sarcoma. Such tumors are not well circumscribed; they grow rapidly, invading the adjacent connective tissue, bone, and lymphatics. Occasionally, the gingiva is the site of metastatic tumors expanding from a central bony site. Also, the enlargement of the gingiva in leukemia brings the patient to the attention of the dentist.

The following tumors and tumor-like growths shall be considered in this discussion:

Benign, epithelial: papilloma, ameloblastoma.
Benign, connective tissue: fibroma, myxofibroma, lipofibroma, ossifying fibroma, osteoma, neurilemmoma, neurofibroma, congenital myoblastoma, hereditary

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fibromatosis gingivae,* hereditary fibromatosis of tuberosity,* congenital fibroadenoma, dilantin sodium hyperplasia,* hemangioma, epulis of chronic hyperplastic gingivitis,* hormonal hyperplasia of pregnancy,* pyogenic granuloma,* giant cell reparative granuloma,* solitary plasmacytoma, eosinophilic granuloma.

Malignant, epithelial: squamous cell carcinoma.

Malignant, connective tissue: fibrosarcoma, leukemia.

BENIGN TUMORS

Papilloma. The papilloma represents the benign tumor of the epithelium. The epithelium proliferates and is disposed in folds with the connective tissue. This causes the growth to be elevated above the surface of the gingiva in a manner similar to the verruca vulgaris or wart. The lesion is sessile and pale, with a rough horny surface. An unusual amount of keratin or parakeratin may be trapped in the folds of epithelium. The connective tissue shows moderate vascularity and considerable chronic inflammation.

Another variety of the papilloma generally resembles the verruca vulgaris. It has a long branching connective tissue core resembling a tree. It is attached to the gingiva by a stalk and resembles a mushroom or cauliflower. This tumor usually is single whereas the verruca vulgaris may be multiple. There may be acanthosis and rarely dyskeratosis with malignant degeneration. However, it is significant that although the papilloma may be quite large, it requires only a moderate amount of blood supply to nourish its cells. This is an index of the benign nature of the tumor.

Ameloblastoma. The rare peripheral ameloblastoma is an odontogenic tumor arising in the oral epithelium. Robinson[8] described such a tumor arising on the lingual side of the ramus without invasion of the bone. It was composed of squamous, glandular and enamel organ epithelium. The oral epithelium has odontogenic properties which may give rise to the ameloblastoma. Such peripheral tumors are considered by some investigators to be basal cell carcinomas. However, the presence of calcified odontomes developing in conjunction with the ameloblastoma suggests odontogenic tumor. Such tumors will invade the bone, causing enlargement of the mandible with considerable destruction of bone.

Fibroma. The fibroma arises from the deep connective tissue, periosteum and periodontium. It is of firm to hard consistency, pale pink in color and usually sessile. Such tumors are slow growing and may be present for many years before treatment is sought. The surface epithelium is usually keratinized but, as it is subject to injury, the tumor

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* These tumor-like growths are not true neoplasms but are included for purposes of discussion.

surface may be red or ulcerated (Fig. 1). The fibroma may be very cellular, showing dense accumulation of plump fibroblasts and small quantities of collagen fibers. Such a tumor is soft, clinically. With time the continued apposition of collagenous fibers causes the fibroblasts to flatten and disappear. This tumor is hard (Fig. 2). Although the etiology of fibromas is not known, reparative granulomas which resolve by fibrosis resemble the fibroma both clinically and histologically.

Myxofibroma. The fibroma may show variation by areas of stellate cell production in a mucoid matrix mixed with the dense collagenous
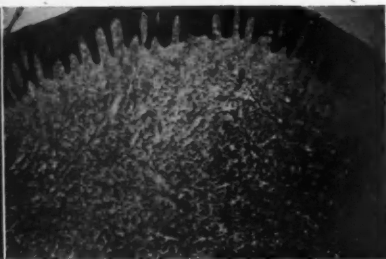
Fig. 1.   Fig. 2.

Fig. 1. Fibroma of the gingiva. The surface is smooth and red.
Fig. 2. Fibroma showing regular epithelium covering dense bundles of collagenous fibers.

fibers. Clinically such tumors arise in the alveolar mucosa and are soft, nodular, and grey in color. The immature stellate cells indicate either a degeneration of the fibroma or the possibility of sarcomatous change. Fibromas exhibiting rapid growth and invasion of the bone usually show myxomatous changes.

Lipofibroma. Fat has its origin from mesenchyme. Occasionally the fibroma will show varying numbers of mature fat cells. Also, there may be found young and undifferentiated connective tissue cells (lipoblasts) showing minute droplets of fat in the cytoplasm. Such fat cells are held in the dense stroma of the fibroma. The lipofibroma may be found in the palatal gingiva of the premolar-molar area and measure 1 cm. in diameter. Such tumors are firm to soft in consistency and show a yellow cast.

Ossifying Fibroma. The most frequent variation of the fibroma results from the change of some fibroblasts to osteoblasts with the production of immature repair bone, dense bone or cementum. Frequently concentric apposition of calcified substance occurs in the cementing substance of the fibroma. The presence of mature bone in a

fibroma removed from the gingiva is not unusual. Such tumors are slow growing, hard, and pink in color.

Osteoma. The periosteum may produce small or large slow-growing, solitary, bony-hard tumors composed of dense cortical bone or cancellous bone. The overlying mucosa is thin and blanched pink in color. They may grow to large size, displacing the adjacent teeth and filling in the palate, vestibule, or floor of the mouth.

Frequently there arise on the labial gingiva of the maxilla and mandible multiple small bony elevations measuring 2 to 4 mm. in diameter. The overlying mucosa is blanched pink in color. Such enlargements arise as subperiosteal cortical bone overgrowths. While they are histologically similar to osteomas they are not considered as neoplasms. They present a problem in edentulous jaws when denture construction is considered.

Neuroma. The neurogenic tumor arises on the gingiva as a firm, light pink, sessile mass. Occasionally it grows to a large size and undergoes degeneration and becomes soft. Such tumors arise in the sheath or terminus of a peripheral nerve. The *neurilemmoma* is a solitary encapsulated benign tumor composed of Schwann cells with whorling bundles of collagenous fibers, palisading wavy nuclei and delicate cleft-like capillaries. The *neurofibroma* may be single or multiple and the proliferation of Schwann cells, collagen production, goes on within the nerve sheath, causing the nerve to expand and become tortuous.

Such tumors are found in great numbers covering the skin and oral mucosa in von Recklinghausen's disease. The tumors vary in size from a few millimeters to several centimeters and have a soft consistency. The skin shows pigmentation or cafe au lait color. Sarcomatous degeneration may occur in one or more such tumors.

Congenital Myoblastoma. There are some interesting congenital tumors of the gingiva. The congenital myoblastoma or congenital epulis, as it is sometimes called, appears at birth. It may be attached to the gingiva by a stalk or it may be sessile. It grows to the size of a pea, is soft in consistency, and is red in color. It was believed that misplaced myoblastic cells proliferate and form a tumor on the gingiva. However, Custer and Fust[4] suggested that this tumor is a degenerating neurogenic tumor. Histologically, there are large polyhedral cells with eosinophilic granular cytoplasm, round uniformly staining nuclei and a fibrous stroma. In morphology the cells strongly resemble true xanthomas although they do not contain lipoid material.

Hereditary Fibromatosis Gingivae. Fibromatosis of the gingiva is a rare enlargement characterized by diffuse dense fibrous hyperplasia of the gingiva beginning early in life. The interdental papillae appear

as multiple fibromas, light pink in color, with a stippled surface and of hard consistency (Fig. 3). The slow, progressive growth may completely cover the crowns of the teeth. Histologically, there are long pointed epithelial ridges covering dense masses of collagenous fiber bundles. There is a striking resemblance to Dilantin sodium hyperplasia of the gingiva.

Fibromatosis of Tuberosity. The maxillary tuberosities may show progressive fibrous enlargement causing a widened mass. The en-

Fig. 3. Fibromatosis gingivae. Note pale, firm, fibroma-like masses.
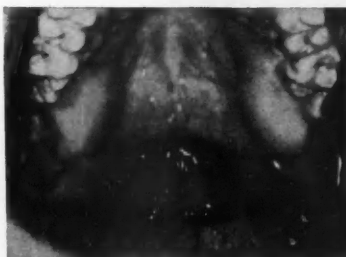
Fig. 4.   Fig. 5.

Fig. 4. Fibromatosis of tuberosities. Note bilateral bulbous expansions.
Fig. 5. Fibromatosis of tuberosities. Dense collagenous fiber bundles with absence of inflammation.

largements may be either movable and pendulous or fixed, pink in color, and of firm to hard consistency. They may be in contact with the opposing teeth or retromolar triangle of the mandible. Frequently, the maxillary third molars are completely covered by the growths (Fig. 4). The growths show a familial tendency; the patient in Figure 4 had a sister with similar enlargements of the tuberosities. Histologically, the tumor consists of very dense bundles of collagenous fibers showing no inflammation and covered by normal stratified squamous epithelium (Fig. 5).

Congenital Fibroadenoma. An unusual gingival tumor present since birth is a slow-growing, pink, firm, irregularly nodular mass of gingiva in the maxillary anteriors (Fig. 6). The histologic appearance is one of dense fibrous bundles with moderate round cell infiltration. Long strands of uniformly staining cells of a salivary adenoma were seen between the bundles of collagen. This was diagnosed as a fibroadenoma. Frequently epithelial tumors show an unusual desmoplastic activity inducing an overproduction of collagen and thereby incarcerating the tumor cells.

Dilantin Sodium Hyperplasia. Some patients treated for epileptic convulsive seizures with sodium diphenyl hydantoin (Dilantin sodium) show a progressive enlargement of the gingiva which may cover

Fig. 6. Congenital fibroadenoma.

the entire dentition on the buccal and lingual surfaces. There are two conditions present in patients who exhibit the enlargement. One is the presence of a chronic inflammatory exudate; the other is the presence of the drug. Somehow, the drug enhances the repair activity associated with the inflammatory process. It may be that such growths are manifestations of an allergic reaction to the drug. Histologically, there is keratinized stratified squamous epithelium with long epithelial ridges showing bulbous expansions covering dense bundles of collagenous fibers. Round cell infiltration is found between the collagen fiber bundles.

Hemangiomas. Hemangiomas are vascular tumors of the gingiva which may be flat and flame red or elevated, compressible and reddish blue in color. These tumors are present at birth or shortly afterward. Such congenital vascular nevi are considered simply as redundant normal tissue. In Sturge-Weber disease there is a nevus flammeus, or elevated hemangioma, of the skin in the region of the mental foramen, with hemangiomas along the branches of the trigeminal nerve. The patients have convulsion of the contralateral side and meningeal hemangiomas and calcifications on the homolateral side. Figure

7 shows such a lobular, red hemangioma arising in the mental fora-
men. Histologically, mature capillaries with well developed adven-
titia are found.

Epulis of Chronic Hyperplastic Gingivitis. In adults the accumula-
tion of calculus around the teeth may cause a single granulomatous
growth ranging to several centimeters in diameter. Any singular
growth on the gingiva may be termed an epulis, which means "upon
the gum." Such tumors are red, very lobular and show blanched areas
undergoing fibrosis (Fig. 8). They are found as part of the process of

Fig. 7. Hemangioma arising in the mental foramen.
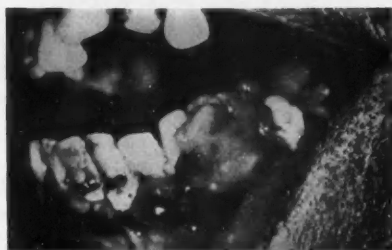
Fig. 8. Epulis of chronic hyperplastic gingivitis.

chronic periodontitis. Histologic findings show mature capillaries,
plasma cells, fibroblasts, fibrosis and edema. It is the quantity of
granulation tissue that characterizes this epulis.

It has been demonstrated by Menken[6] that inflammatory exudate
contains a growth-promoting substance which stimulates epithelial
and connective tissue proliferation. This is interesting, since the
growth-promoting substances come from injured cells. Carrel[2] de-
scribed the presence of growth-promoting substances (trephones)
liberated by cells in a tissue culture. Also, he noted that wound heal-
ing was delayed in aseptically clean wounds but accelerated by the
presence of irritants.

Steroids such as adrenal desoxycorticosterone acetate (DOCA) and

the pituitary growth-promoting substance (STH) promote the inflammatory response and repair.[9,10] Antagonistic steroids cortisone and ACTH inhibit the inflammatory response and repair.[7,9] It is significant that although DOCA and STH are antagonistic to cortisone and ACTH, the former always exert the dominant influence in repair and inflammation. Estrogens have been shown to promote connective tissue fiber formation and then inhibit such formation.[5] This is thought to be due to inhibition of pituitary STH production by the estrogens.[9] The significance of this discussion is important in understanding that the growth of those oral tumors which are fundamentally inflammatory reparative granulomas is under hormonal control.

Fig. 9. Pregnancy tumor.

The budding of capillaries, the proliferation and differentiation of fibroblasts and osteoblasts, and the production of collagenous fibers and osteoid tissue may show various disturbances. One may be the vascular overproduction and variation in the capillary size noted in the hormonal hyperplasia of pregnancy and in the pyogenic granuloma. Another is the fibroblastic overproduction and giant cells seen in giant cell reparative granuloma. Such overgrowths show fibrosis as the final product. Frequently, ossification is found in such growths, producing an ossifying fibroma.

Hormonal Hyperplasia of Pregnancy. Tumorous enlargements showing a preponderance of vascular tissue are seen in women, most often during the second trimester of pregnancy. A single interdental papilla may show progressive enlargement to 1 cm. or more, demonstrating nodulation and a raspberry redness (Fig. 9). There are loss of stippling and a tendency toward hemorrhage. Such a mass may cover the teeth. Frequently, such gingival growths are multiple, affecting the marginal and interdental gingiva (hormonal gingivitis of preg-

nancy). Histologic examination shows numerous vascular channels, some of sinusoidal proportions, and diffuse infiltration by plasma cells and polymorphonuclear neutrophils. There usually is edematous connective tissue; however, fibrosis is frequently observed.

Pyogenic Granuloma. The pyogenic granuloma is a singular vascular growth associated with local injury by sharp bony spicules or retained root tips showing suppuration; it also occurs about teeth involved in periodontitis. It is red, lobular, soft, hemorrhagic and painless. Such growths were thought to be due to pyogenic infections, but an infection is only one of many injurious agents stimulating inflammation and repair. The vascular channels and inflammatory exudate are identical with that found in pregnancy tumor. A pyogenic granuloma observed in a 40 year old man is shown in Figure 10.

Fig. 10.   Fig. 11.

Fig. 10. Pyogenic granuloma. Smooth, red, soft, hemorrhagic surface.
Fig. 11. Giant cell reparative granuloma.

Giant Cell Reparative Granuloma. The giant cell reparative granuloma represents the fibroblastic preponderance in overgrowths of the repair tissue. The presence of multinucleated giant cells characterizes the lesion. This growth is highly vascular and shows hemorrhage and blood pigment within its mass. The lesion has enjoyed a popularity among those found on the gingiva and had become synonymous with the term epulis, although in its broad sense that term applies to any benign tumor of the gingiva. It must be pointed out that this tumor represents only another form of overgrowth of repair tissue and that it may be found in the dental granuloma, cyst wall, epulis fissuratum, centrally in the jaw bone, and it has been reported in granulations in surgical wounds of the face.

The growth is found more frequently in the mandible than in the maxilla, and in the region of the first molar area more often than in the anterior gingiva. It arises rapidly in an area of inflammation or

following a history of injury, grows rapidly and then stops. It appears reddish blue in color and is firm in consistency, with a smooth to lobular surface. It may cause a diastema with the mass on the labial and lingual surface (Fig. 11) and it may erode the bone, displacing the teeth and filling in the vestibule. Histologically there are a node of young fibroblasts densely packed together and frequent multinucleated giant cells with fifteen to thirty centrally disposed nuclei, adja-
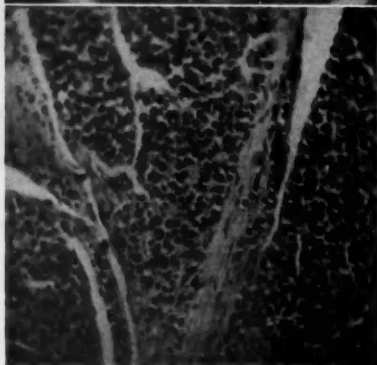
Fig. 12.

Fig. 13.
Fig. 12. Solitary plasmacytoma.
Fig. 13. Plasmacytoma. Note dark eccentric nuclei and delicate capillary cleft.

cent to a capillary slit. Hemorrhage and blood pigment may be found. There is dense fibrous connective tissue around the node. In time, the node is replaced by fibrous tissue, again indicating the benign functional nature of this growth.

Plasmacytoma. A painless, red, firm tumor, the solitary plasmacytoma, may be found in the oral cavity, pharynx, and maxillary sinus. This tumor, which has been observed to arise upon the gingiva, has a red-blue shiny mucosa, a firm consistency, and a sessile base (Fig. 12). The tumor appears suddenly and grows rapidly, demonstrating some

erosion of cortical bone. The histologic picture is one of densely packed plasma cells with eccentric nuclei. There appears to be some variation in the nuclear size and staining. There is a delicate reticular stroma with thin single-cell vascular slits and slight collagenization (Fig. 3). Such tumors, although generally classed as benign, have shown invasive ability and may be malignant. Histogenetically, this tumor is related to the reticulo-endothelial system.

Fig. 14.
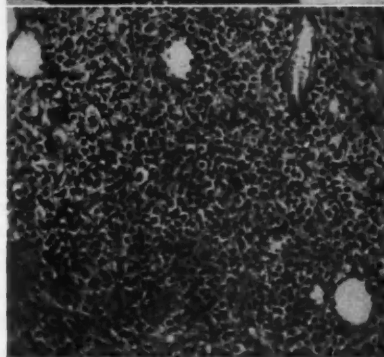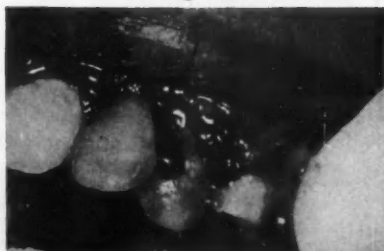
Fig. 15.

Fig. 14. Eosinophilic granuloma.
Fig. 15. Eosinophilic granuloma. Note numerous polymorphonuclear eosinophilic leukocytes. The histiocytes have centrally placed nuclei and clear cytoplasm.

Eosinophilic Granuloma. A granulomatous lesion with tumorous and erosive characteristics which may be found on the gingiva is the eosinophilic granuloma, although this lesion is observed principally within the bone marrow of flat bones and long bones. However, numerous solitary skin and mucous membrane lesions have been reported. The gingival lesion appears red, lobular, and erosive and causes loss of bone and exposure of the root of the tooth. Frequently, the lesion begins about the root of a tooth and erodes outward to the gingiva (Fig. 14). Roentgenographic examination may show irregular radio-

lucent areas resembling dental granulomas. The histologic picture is one of a granuloma. There are many dilated capillaries with swollen endothelial cells, and many histiocytes with clear or foamy cytoplasm and dark, round, centrally placed nuclei. The histiocytes usually are filled with cholesterol. Also present in varying quantities are polymorphonuclear and binuclear eosinophilic leukocytes (Fig. 15). The lesions are benign and may be treated surgically. However, the wide distribution of the lesions, so frequently observed in the bones, makes radiation the treatment of choice.

MALIGNANT TUMORS

Squamous Cell Carcinoma. The gravest disease of the oral cavity is the squamous cell carcinoma. It constitutes more than 90 per cent of the oral malignancies. It has been estimated that 5 per cent of human cancers occur in the mouth.[3] In a study of 401 cases of oral carcinoma, Bernier[1] reported that 12 per cent of the oral carcinomas arose on the gingiva.

It is well established that oral leukoplakia may be a premalignant lesion. This disturbance is more frequently observed on the buccal mucosa and tongue than the gingiva, however. It is evident that chronic irritation predisposes to leukoplakia and carcinoma. Studies of smoking cigarettes, pipes, and cigars have shown that the burning coal is at 400° C. This high temperature may fractionate hydrocarbons in the tobacco and produce carcinogens in the smoke. The smoke as it enters the mouth is at about 37° C.

Cancer of the gingiva may grow and invade the underlying bone and completely erode the mucosa, exposing the roots of the teeth (Fig. 16). The mucosa is pink, lobular, and firm, with a red eroded margin. Such deep ulcers appear as advanced periodontal disease. Roentgenographic examination shows that the bone is absent, and the roots of the teeth appear to be floating in the radiolucent space. The teeth are loose and usually painless; however, deep aching pain may be associated with such lesions. The outgrowing, exophytic, spreading, gingival carcinomatosis creates a lobular, pink, irregular, diffuse tumor of the gingiva. Irregular patches of hyperkeratosis are found over the tumor surface (Fig. 17). Direct invasion of the bone creates widened marrow spaces as the bone resorbs in response to the proliferating tumor. In the mandible the tumor may spread along the mandibular canal. The bone presents an irregular radiolucent area with diffuse margins upon roentgenographic examination. Invasion of bone creates difficult treatment problems requiring radical operative procedures. The tumor may reach the regional submental,

submaxillary, and cervical lymph nodes by permeation and emboli-
zation from the lymphatics. Such nodes are stony hard and painless.
This finding also indicates a poor prognosis and requires radical oper-
ative procedures.

Fibrosarcoma. Fibrosarcoma of the gingiva may arise from either
the periosteum or the cervical periodontium. The tumor develops
rapidly, with the fibroblasts showing considerable proliferative activ-
ity but little collagen fiber production. Fibroblasts which do not have
time to mature in the intermitotic phase may go on to another mi-
totic phase without producing collagenous fibers. Clinically, the fibro-
sarcoma grows to large size with a broad base and is of firm con-
sistency. The surface is lobular and shows ulcerations with hemor-
rhage. The teeth become loose and displaced, and pain is usually

Fig. 16.   Fig. 17.

Fig. 16. Squamous cell carcinoma eroding the gingiva and palate, exposing
the roots of the teeth.
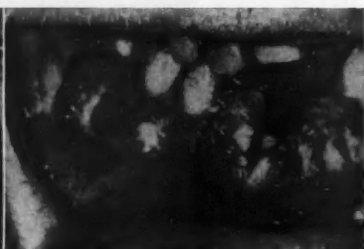
Fig. 17. Squamous cell carcinoma. Note hyperkeratotic plaques on the tumor
masses.

present. This tumor frequently occurs in children and young adults.
Its tendency to early metastasis via the blood stream creates an un-
favorable prognosis. Fortunately, this tumor is rare.

Leukemia. The gingiva undergoes enlargement in the leukemias.
Leukemia is a malignant overproduction of white blood cells of hemo-
poietic or lymphoid tissues. The classification includes acute and
chronic myelogenous, monocytic, and lymphatic leukemias.

Acute leukemia affects children most frequently and is rapidly
fatal. The white blood count may be over 100,000 per cubic milli-
meter with a high percentage of immature forms. These circulating
cells infiltrate all the organs of the body including the gingiva. Often
the first complaint may be of oral bleeding or infection. The gingiva
appears swollen, red-blue in color, and hemorrhagic, resembling
chronic hyperplastic gingivitis. Frequently, there is necrotizing ulcer-
ative gingivitis characterized by severe pain, ulcerations and necrosis

of the gingiva. The patients are febrile, and show regional lymph-adenopathy and subcutaneous and submucous petechiae. The bleeding and clotting times are prolonged, and anemia is usually present. No oral surgical procedures should be undertaken in acute leukemia. Figure 18 shows the gingiva in a case of acute myelogenous leukemia in a woman 26 years of age; she had a white blood count of 115,000, and myelocytes and myeloblasts were the preponderant cells. Note
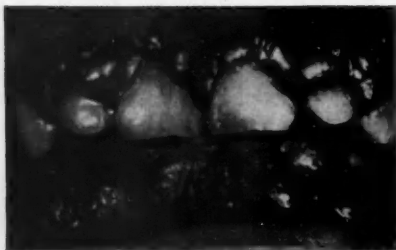
Fig. 18. Acute myelogenous leukemia. Note hyperplastic gingiva.

the hyperplastic papillae and hemorrhagic exudate. The color was magenta and the consistency was turgid.

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Benign Tumors and Cysts of the Jawbones

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The benign tumors of the jawbones to be considered in this section comprise a group of lesions all derived from the cellular components of bone, cartilage and other associated connective tissue elements including blood vessels and nerves. Some of these lesions are tumors only in the broadest sense of the word since they produce an obvious tissue mass but occur as a result of inflammation and are not actually neoplastic.

There are many other benign tumors which may occur centrally within the jaws but they are chiefly the lesions associated with the odontogenic apparatus and, with the exception of the odontogenic cysts, will be considered in a separate article.

FIBRO-OSSEOUS LESIONS OF THE JAWS

The jaws may be the site of development of a gamut of central lesions which present varying clinical, roentgenographic and histologic characteristics but which have a common origin from fibro-osseous tissue. The relationships of these various lesions are only gradually being appreciated and a reconciliation of views concerning their true nature can be predicted in the near future.

Similar lesions occur, with few exceptions, in other bones of the body so that these cannot be considered native only to the jaws. The peculiar anatomic and physiologic characteristics of the jaws provide an unusual opportunity for unique reactions of the tissues to various stimuli not shared by other bones. To this may be attributed some of the peculiarities of this group of lesions.

The fibro-osseous lesions of the jaws to be discussed here include those commonly described under the following terms: (1) fibrous dysplasia of bone, (2) central giant cell reparative granuloma and giant cell tumor, and (3) central fibroma of bone.

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Fibrous Dysplasia of Bone

Fibrous dysplasia of bone is one of the most perplexing diseases of osseous tissue to confront the dentist. The disease may affect many bones in a single individual (polyostotic form) or only a single bone (monostotic form). The former is exceptionally rare and is frequently associated with skin pigmentation and, in some instances, with endocrine disturbances.

Monostotic fibrous dysplasia of the jaws has been described under many names including fibro-osteoma, ossifying fibroma, localized osteodystrophy, osseous dysplasia and leontiasis ossea as well as others. It presents no systemic manifestations.
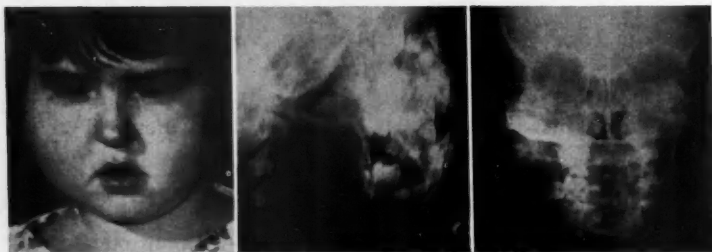
Fig. 1. Fibrous dysplasia of bone.

Etiology and Pathogenesis. The etiology of fibrous dysplasia is unknown although a variety of possible factors has been suggested without universal agreement. There is general belief that the lesion represents a developmental or dysplastic malformation of bone, not a true neoplasm, while some evidence suggests that local infection or trauma may be the cause of this disease. Regardless of its cause, the course of development is essentially an alteration in the direction of normal bone development with proliferation of fibroblasts, production of excessive amounts of connective tissue and irregular formation of bone.

Clinical and Roentgenographic Features. Monostotic fibrous dysplasia generally occurs in young persons and has no definite predilection for either sex. The first clinical sign of the disease is usually a painless swelling of the jaw although the patient may complain of vague discomfort (Fig. 1). Some malalignment or displacement of teeth may occur owing to the progressive expanding nature of the lesion.

The roentgenographic appearance of fibrous dysplasia is not pathognomonic. It appears as an irregular radiolucency which may or may not exhibit definite loculations (Fig. 1). Some lesions present a patchy radiopaque appearance, depending upon the amount of bone

formation present. The periphery may be either smooth or ragged. The cortical bone appears greatly thinned but the outer plates of bone are seldom perforated. Little periosteal proliferation of bone is present.

There is considerable microscopic variation between cases of monostotic fibrous dysplasia of the jaws. Essentially, the lesion is made up of a fibrillar matrix interspersed with numerous proliferating fibroblasts, and usually there are scattered irregular trabeculae or foci of ossification throughout the lesion. Variable numbers of multinucleated giant cells are often present.

Treatment and Prognosis. The treatment of fibrous dysplasia consists of surgical excision or curettement of the tumor-like mass. The tissue may or may not separate easily from the surrounding bone, and the lesion seldom recurs.

Familial Fibrous Dysplasia

This unusual form of fibrous dysplasia, sometimes called "cherubism," is a disease of the jaws which appears in early childhood and often affects several members in one family.

Clinical and Roentgenographic Features. Familial fibrous dysplasia of the jaws usually manifests itself by the age of three to four years as a progressive, firm symmetrical swelling of the maxilla or mandible producing a typical cherubic facial appearance. The deciduous dentition may be shed prematurely and the permanent dentition may be deficient in number. The disease itself is usually painless.

Roentgenograms reveal bilateral central destruction of the jaws with expansion and thinning of the cortical plates. The bone often appears multilocular. The other bones of the body including the skull are not involved.

The histologic appearance of the lesion varies from case to case but in general is similar to that of monostotic fibrous dysplasia.

Treatment and Prognosis. Most cases of familial fibrous dysplasia, although progressing quite rapidly during early childhood, tend to become static and even show regression as the patient reaches puberty. Surgical treatment is usually not feasible because of the widespread nature of the disease. After puberty, cosmetic surgery may be carried out.

Central Giant Cell Reparative Granuloma

The central giant cell reparative granuloma of the jaws has also been called a central giant cell tumor. Some investigators believe that

two distinct lesions exist, one a granuloma and the other a neoplasm, but the criteria for their separation are not clearly established. Giant cell neoplasms of other bones are well recognized and in many instances these act very aggressively. It is only reasonable to assume that analogous lesions would occur in the jaws. Rare lesions, characterized by the presence of many giant cells, do destroy bone and exhibit vigorous growth activity and these may represent a true neoplasm. They should not be confused with the giant cell tumor of hyperparathyroidism.

Etiology and Pathogenesis. Trauma to the bone appears to be one of the most significant etiologic factors in the development of the central giant cell reparative granuloma. Exactly why the tissues respond with this particular reaction is not certain but may relate to the intramedullary hemorrhage with subsequent mobilization of multinucleated giant cells to act as phagocytes.

Clinical and Roentgenographic Features. The lesion occurs chiefly in persons younger than 20 years, with equal predilection for the maxilla and the mandible. The patient complains only of a swelling of the jaw although there may be some local discomfort also. Tooth displacement occasionally occurs.

The roentgenographic appearance of the central giant cell reparative granuloma is one of diffuse bone destruction with expansion and thinning of the cortical plates. The lesion may appear multiloculated or present a honeycombed pattern. The microscopic appearance of the lesion is one of numerous multinucleated giant cells scattered throughout a vascular fibrillar stroma with evidence of focal hemorrhage.

Treatment and Prognosis. This lesion is treated by surgical excision although its borders are often not well demarcated. Despite the inflammatory nature of the condition, recurrence may follow inadequate removal.

Central Fibroma of Bone

The central fibroma of bone is a rare lesion of the jaws composed entirely of fibrous connective tissue with or without foci of calcification or ossification. The tumor occurs at any site, has no apparent age predilection, and manifests itself as a slowly growing painless expanding lesion. The roentgenogram usually reveals a well circumscribed unilocular radiolucency with foci of opacity if calcification is present. The cortical plates may be thin and expanded. Differentiation of this fibroma from fibrous dysplasia is difficult.

Treatment is by surgical excision and recurrence is uncommon.

MYXOMA OF BONE

The myxoma, a rare benign neoplasm resembling primitive mesen-
chyme, arises both in soft tissues and centrally within various bones.
The intrabony tumor occurs at any age and involves either the max-
illa or the mandible, without apparent predilection. This myxoma is
usually an infiltrating, slowly growing, painless lesion which produces
destruction of bone as well as expansion of the outer plates.

The treatment is surgical excision. Recurrence is relatively com-
mon but malignant transformation with metastasis is rare.

HEMANGIOMA OF BONE

The hemangioma is a common benign tumor characterized by the
proliferation of blood vessels. Most workers believe that this is not a

Fig. 2. Hemangioma of bone.

true neoplasm but rather a vascular malformation or hamartoma. It is
often congenital and follows a benign course, frequently undergoing
spontaneous regression.

Central hemangiomas of bone, including those of the maxilla and
mandible, occur with some frequency and may present difficulty in
diagnosis. There is seldom any clinical evidence of the presence of
such a central lesion, particularly when small, although pain may be
a symptom. Larger lesions frequently expand the bone and produce
obvious facial asymmetry. The roentgenogram reveals a destructive
lesion of bone, sometimes with a honeycombed appearance, but with
no especially distinctive features. The proximity of the radiolucent
area to the apices of teeth has given rise to the erroneous diagnosis of
periapical involvement (Fig. 2), and extraction of the teeth has been
followed by profuse bleeding resulting in death in at least one re-
ported case.

Treatment of the central hemangioma consists of ligation of the main artery supplying the tumor followed by surgical excision of the vascular mass. Injection of a sclerosing solution has also been used.

CHONDROMA

The chondroma is a benign neoplasm of cartilage which is uncommon in the jaws. It develops as a painless, slowly expanding lesion with no definite age predilection. Vestigial cartilaginous rests, present in both the maxilla and the mandible, seem to enhance the possibility of development of the chondroma. The roentgenogram shows an irregular destructive lesion which may cause resorption of roots of teeth in the area of involvement.

The chondroma is treated by surgical excision, with recognition that this tumor tends to undergo malignant transformation in some instances, even after remaining quiescent for relatively long periods of time.

OSTEOMA

The osteoma is a benign neoplasm characterized by either endosteal or periosteal proliferation of bone. In the jaws, it is difficult to
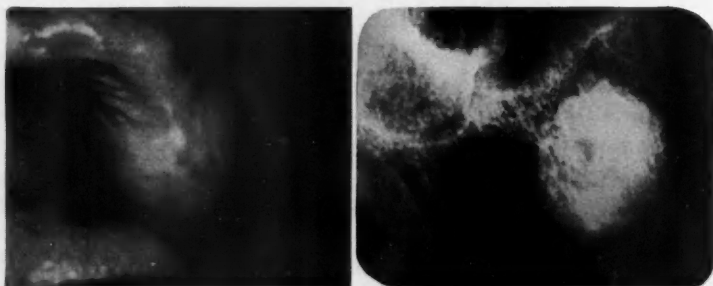
Fig. 3. Osteoma.

differentiate between a true neoplasm of bone and proliferation of bone on the basis of irritation or infection. Sclerosis of bone in the jaws due to a low grade infection is extremely common. The true osteoma may be confused also with the exostosis and enostosis.

The osteoma is not a common oral lesion. It seems to occur at any age in either jaw as a very slowly progressive, painless lesion which may produce some expansion of the outer plates of bone (Fig. 3). The lesion appears on the roentgenogram as a well circumscribed, dense opaque mass and upon microscopic examination as heavy, compact bone.

Treatment of the osteoma is surgical removal. This is sometimes deferred until the lesion begins to expand or until a prosthetic appliance is to be constructed. In this latter event, removal of the tumor is recommended particularly when it lies close to the surface of the alveolar bone. The lesion does not tend to recur.

OSTEOID OSTEOMA

The osteoid osteoma is a benign tumor of very rare occurrence in the jaws. Its true nature is uncertain but has been suggested to be either a true neoplasm or an unusual inflammatory reaction of bone. The lesion occurs chiefly in young persons and first manifests itself by severe pain and occasionally swelling of the bone. The roentgenogram presents a unique, almost pathognomonic, picture of a small, round or ovoid radiolucency surrounded by a rim of sclerotic bone. The central radiolucency may show evidence of calcification. The entire lesion seldom attains a size greater than 1 cm. in diameter.

Treatment is surgical excision; the lesion does not recur.

TRAUMATIC NEUROMA

The traumatic or amputation neuroma is not a true neoplasm but rather a hyperplasia of nerve tissue occurring as a result of an exuberant attempt at repair of a damaged nerve trunk. It is a rather common sequela of accidental or purposeful sectioning of a nerve and may follow tooth extraction.

Etiology and Pathogenesis. Severance of a nerve is followed by degeneration of nerve fibers with disintegration of the axis cylinders and myelin sheaths. The nerve does not disappear completely, however; the neurilemmal sheaths persist and it is through these that new fibers proliferate.

If the proliferating proximal nerve meets some obstruction such as scar tissue or malaligned bone following a fracture, actual reinnervation of the distal part does not occur. Instead there may be continued proliferation of the proximal nerve end into a bulbous or nodular mass of nerve fibers and cells of the nerve sheath, forming a neuroma.

Clinical and Roentgenographic Features. The traumatic neuroma occurs frequently near the mental foramen, in association with a tooth socket such as the mandibular third molar after a difficult extraction or associated with the mandibular nerve at any point along its course, as well as in soft tissues such as the tongue and lip. It is a slowly growing lesion which may be painful if situated near the surface and if subjected to pressure.

The roentgenogram may reveal a well circumscribed radiolucent area associated with the course of a nerve, but otherwise there is nothing distinctive to suggest the presence of a neuroma.

Treatment and Prognosis. Surgical excision of the traumatic neuroma is the treatment of choice because of the associated pain and progressive nature of the lesion. Recurrence is not common, particularly if a small section of the proximal portion of the involved nerve is also removed, even though the treatment is similar to the original injury which preceded the development of the lesion.

NEURILEMMOMA

The neurilemmoma is an uncommon neoplasm derived from the nerve sheath. It is more common in soft tissues but has been reported to occur centrally within the jaws. In these cases, it causes destruction of bone and often remarkable expansion of the jaw. There may be some pain associated with the tumor.

The roentgenogram is not distinctive and exhibits a circumscribed radiolucency which sometimes appears multiloculated. The diagnosis can only be established by biopsy.

Treatment consists of surgical removal; the neoplasm is usually encapsulated. This lesion may recur following incomplete removal but it does not exhibit the propensity for malignant transformation that is sometimes seen in the neurofibroma.

DEVELOPMENTAL (FISSURAL) CYSTS

Developmental or fissural cysts of the jawbones are cysts, not associated with the dental apparatus, which develop as a result of proliferation of epithelium entrapped or enclosed within bone along lines of fusion of primitive processes during embryonic life or from vestigial epithelial structures. They occur at certain specific sites within the jaws and, because of this, may present a pathognomonic roentgenographic appearance.

The most common of the developmental or fissural cysts are (1) nasopalatine duct cyst, (2) median palatal cyst, and (3) globulomaxillary cyst.

Nasopalatine Duct Cyst

This cyst is also called an incisive canal cyst or median anterior maxillary cyst and occurs with greater frequency than any of the other developmental cysts.

Etiology and Pathogenesis. The epithelium lining the cyst is de-
rived from the primitive or vestigial nasopalatine ducts which orig-
inally connected the embryonic oral cavity and nasal cavity. The ac-
tual cause for the proliferation of epithelium in this region is un-
known.

Clinical and Roentgenographic Features. The majority of nasopala-
tine duct cysts are asymptomatic and are discovered incidentally dur-
ing routine roentgenographic examination of the jaws. Sometimes
the cyst becomes infected and may perforate the oral mucosa on or
adjacent to the incisive papilla to drain the suppurative contents of
the sac. The lesion appears on the maxillary anterior roentgenogram

Fig. 4.   Fig. 5.   Fig. 6.

Fig. 4. Nasopalatine duct cyst.
Fig. 5. Median palatal cyst.
Fig. 6. Globulomaxillary cyst.

as a well circumscribed, round, ovoid or heart-shaped radiolucency
between or above the roots of the maxillary central incisors (Fig. 4).
A border of sclerotic bone is sometimes seen surrounding the lesion.
Care must be taken not to confuse a large incisive canal with an ac-
tual cyst in this location.

These cysts may be lined by either stratified squamous epithelium
or pseudostratified ciliated columnar epithelium.

Treatment and Prognosis. Surgical excision is indicated, especially
if the cyst is infected and has established drainage. The lesion does
not recur frequently.

Median Palatal Cyst

The median palatal cyst is a distinct entity although it has often
been confused with the nasopalatine duct cyst.

Etiology and Pathogenesis. The epithelium forming the lining of this cyst is derived from epithelium entrapped during the closure and fusion of the palatine processes in the midline of the palate.

Clinical and Roentgenographic Features. There may be little clinical evidence of the presence of this cyst until it reaches a relatively large size, when a midline bulging of the palate becomes obvious. The overlying mucosa retains its normal color and there is generally no ulceration or perforation of the surface.

The roentgenogram provides a remarkable picture of a symmetrical midline destruction of bone which may be quite extensive but is usually smoothly outlined, indicative of its slowly progressive expansile growth (Fig. 5). The epithelium lining this cyst is usually stratified squamous in type but may exhibit areas of respiratory tract origin.

Treatment and Prognosis. The median palatal cyst should be surgically removed. There is little tendency for recurrence.

Globulomaxillary Cyst

The globulomaxillary cyst is a relatively uncommon form of fissural cyst but should be considered because of its frequent confusion with the periodontal cyst.

Etiology and Pathogenesis. This cyst is derived from epithelium entrapped during the closure and fusion of the globular portion of the medial nasal process with the maxillary process. Thus, it is located usually between the maxillary lateral incisor and cuspid.

Clinical and Roentgenographic Features. The globulomaxillary cyst seldom produces any clinical manifestations of its presence in its early stages of development. As the cyst enlarges, it often causes separation of the lateral incisor and cuspid and may even produce bulging of the labial plate of bone. Occasionally, this cyst will perforate the bone and mucosa, forming a fistulous tract emptying on the surface between these teeth. Unless affected by an unrelated concomitant pathologic process, the adjacent teeth are vital.

The intraoral roentgenogram exhibits a radiolucency, often in the shape of an inverted pear, situated between the incisor and cuspid and causing divergence of the roots (Fig. 6). This area of bone destruction is generally sharply outlined. Difficulty may be encountered in distinguishing this developmental cyst from a lesion associated with periodontal pathosis, lateral or apical.

Treatment and Prognosis. This cyst should be surgically removed, preserving the adjacent teeth if possible. There is little tendency for recurrence of this lesion.

ODONTOGENIC CYSTS

Odontogenic cysts are derived from epithelium which possesses the potentiality for forming dental tissue or which has already formed such tissue and has passed into a quiescent state. The nature of these cysts varies depending upon their stage of development with respect to tooth formation and upon their site.

The most common of the odontogenic cysts are (1) primordial cyst, (2) dentigerous cyst, and (3) periodontal cyst.

Primordial Cyst

This cyst, sometimes referred to as a follicular cyst, is the least common of the odontogenic cysts and arises at an earlier stage of odontogenesis than the other dental cysts.

Fig. 7.   Fig. 8.

Fig. 7. Primordial cyst.
Fig. 8. Dentigerous cyst.

Etiology and Pathogenesis. The primordial cyst develops through cystic degeneration of the stellate reticulum of the tooth germ before there has been any opportunity for the formation of calcified tissue. Thus, there is formed an epithelium-lined sac filled with fluid, this epithelium being derived from the inner and outer enamel epithelium.

Clinical and Roentgenographic Features. The primordial cyst seldom presents clinical evidence of its presence inasmuch as it does not attain any great size and exhibits little tendency for expansion of bone. Generally, there is no associated pain.

Its roentgenographic appearance is not distinctive but it is suggested by the occurrence of a well circumscribed radiolucency occurring in place of a tooth when there is no history of a tooth extraction in this area (Fig. 7). Lacking an adequate history relating

to previous tooth extraction, it is impossible to differentiate between a primordial cyst and a residual periodontal or dentigerous cyst. Occasionally, a primordial cyst develops in the place of a supernumerary tooth.

The epithelium lining the primordial cyst is stratified squamous in type and shows varying degrees of proliferation. Inflammatory cell infiltration may be present in the connective tissue wall of the lesion.

Treatment and Prognosis. The treatment for the primordial cyst is surgical removal. There is little tendency for recurrence.

Dentigerous Cyst

The dentigerous cyst, which has also been called a follicular cyst, is a relatively common odontogenic cyst. It is associated with a formed tooth, since it arises at a later stage of odontogenesis than the primordial cyst.

Etiology and Pathogenesis. This cyst appears to originate through alteration of the reduced enamel epithelium with accumulation of fluid either between the layers of the enamel epithelium or between this epithelium and the tooth crown. The dentigerous cyst, therefore, is always associated with the crown of an impacted or unerupted tooth. It may surround the coronal portion of the tooth or may lie lateral to the crown. A dentigerous cyst may occasionally be associated with an odontoma.

Clinical and Roentgenographic Features. The small dentigerous cyst presents no clinical evidence of its presence. As it increases in size, expansion of the bone may occur with ensuing facial asymmetry. The teeth with which this cyst is most commonly associated are the maxillary cuspid and the mandibular third molar. Seldom does this type of cyst perforate to form a fistulous tract although infection in this cyst is commonly seen.

A radiolucent area of variable size partially or completely surrounds the crown of an unerupted tooth (Fig. 8). The periphery of this radiolucency is usually smooth and may show a thin border of sclerotic bone. On occasion, this cyst may cause extensive destruction of bone, e.g., involving the entire ramus up to the condyle and coronoid process.

The term "eruption cyst" has been applied to that form of dentigerous cyst which is in such a position that it appears to be impeding the eruption of the tooth. Because the small dentigerous cyst roentgenographically resembles an enlarged follicular space, differentiation may be difficult.

The dentigerous cyst is lined by stratified squamous epithelium.

Treatment and Prognosis. Surgical removal is the treatment of choice for the dentigerous cyst. Histologic examination of all dentigerous cysts by a qualified oral pathologist is recommended because of the well recognized occurrence of an ameloblastoma developing in the wall of such a lesion from this odontogenic epithelium. Recurrence following complete surgical removal of the lesion seldom is seen.

Periodontal Cyst

There are two recognized forms of periodontal cyst: the apical or common root end (radicular) cyst and the lateral periodontal cyst.

Fig. 9.   Fig. 10.
Fig. 9. Apical periodontal cyst.
Fig. 10. Lateral periodontal cyst.

The former, usually arising as a result of dental caries and ensuing pulp infection, develops in a periapical granuloma and is so common and well recognized as to warrant no further discussion here (Fig. 9).

The lateral periodontal cyst is relatively rare, is of unknown causation and seldom produces any clinical manifestation of its presence. It does not arise as a result of pulp infection inasmuch as the cyst is found high on the lateral surface of the root in intact teeth (Fig. 10). The source of the epithelium is undoubtedly the epithelial rests of Malassez in the periodontal membrane, but the reason for their proliferation is obscure. The treatment of the lateral periodontal cyst is usually tooth extraction with subsequent examination of the soft tissue, since an early ameloblastoma may produce a similar appearance.

TRAUMATIC BONE "CYST"

The traumatic bone "cyst," also called hemorrhagic cyst, extravasation cyst, solitary bone cyst, and unicameral bone cyst, is not a true cyst inasmuch as the lesion does not contain an epithelial lining. Being a cyst-like cavity, however, it will be considered here for the sake of completeness.

Etiology and Pathogenesis. The most commonly ascribed cause is trauma to the bone resulting in hemorrhage into the marrow spaces and clot formation with subsequent failure of organization. The bone within the area of the hematoma undergoes necrosis owing to the local ischemia and is eventually resorbed, along with the remainder of the blood clot, resulting in a cavity within the bone. It is generally agreed that the lesion tends to enlarge gradually, albeit not indefinitely, despite the fact there is no internal pressure present as judged by the absence of bone reactivity at the periphery of the lesion.

The trauma-hemorrhage etiology does not completely explain all clinical facets of this unusual lesion and, for this reason, other possible sources of origin have been suggested. Our present concept of the traumatic bone cyst indicates that this is a specific lesion of unknown etiology resulting basically from lack of normal repair of damaged bone.

Clinical and Roentgenographic Features. The traumatic bone cyst involving the jaws occurs far more frequently in the mandible than in the maxilla and is most common in the molar region. Younger persons are involved more frequently than elderly individuals and it has been suggested that this may be due to the more delicate nature of the blood vessels in the bone marrow of the young. In some series, males are affected more often than females, the explanation being offered that the male is more prone to suffer from trauma than the female by virtue of his activities.

There are seldom any signs or symptoms produced by the traumatic bone cyst. The majority of cases are discovered during routine roentgenographic examination of the jaw. Despite the fact that the lesion does enlarge, only on rare occasions is expansion of the jaw encountered. Pain is absent and the teeth in the affected area are generally vital unless coincidentally affected by some other pathologic process.

The roentgenogram reveals a well circumscribed radiolucent area in the bone, sometimes at considerable distance from the teeth but at other times situated between the roots of adjacent teeth (Figs. 11, 12). Seldom is there any evidence of bone reaction around the periphery of the lesion. The individual lesions are usually round or

ovoid in shape and vary from a few millimeters to several centimeters in diameter.

Surgical entry into the traumatic bone cyst will reveal a cavity which either is completely empty or contains only a tiny bit of fluid or sometimes shreds of blood clot. The wall of the cavity is usually smooth and may be lined with a thin connective tissue membrane which can be removed only in fragments.

Treatment and Prognosis. Treatment consists of entering the lesion and establishing a blood flow and clot. Once formed, this clot usually

Fig. 11.   Fig. 12.
Fig. 11. Small traumatic cyst.
Fig. 12. Large traumatic cyst.
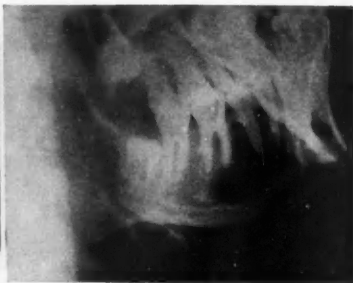
undergoes typical organization and the defect is slowly filled in with bone over a period of several months.

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Odontogenic Tumors

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The term "odontogenic tumors" is generally used to designate a
group of overgrowths which arise from tooth-forming tissues. The
adjective "odontogenic" would seem to imply that these lesions are
tooth-producing tumors, but in many instances this is not the case.
Some members of this group are true neoplasms (uncontrolled new
growths) while others are blastomatoid lesions which generally are
the result of faulty development. The word "tumor" may be applied
properly to all of these entities for it does not necessarily signify a
neoplasm but only an abnormal enlargement.

NEOPLASMS

The odontogenic apparatus is composed of both epithelium and con-
nective tissue. A neoplasm may arise from either or both of these
tissues. The epithelial neoplasm is termed an *ameloblastoma* and the
connective tissue neoplasm is designated an *odontogenic fibroma*. If
both connective tissue and epithelial elements are neoplastic the lesion
is classified as a *fibroameloblastoma*. Occasionally an ameloblastoma
may appear in conjunction with a hemangioma (*hemangioameloblas-
toma*) or with an odontoma (*odontoameloblastoma*). Odontogenic neo-
plasms are primarily benign tumors. A few malignant ameloblastomas
have been reported, but the malignant counterpart of the odontogenic
fibroma (odontogenic fibrosarcoma) is, at present, a theoretical class-
ification.

Ameloblastoma

The ameloblastoma (adamantinoma) is a neoplasm arising from
odontogenic epithelium or from epithelium having the potentiality for

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forming an enamel organ. It may develop from the inner or outer enamel epithelium, from remnants of the dental lamina, from remains of Hertwig's sheath or from the oral epithelium.

The pattern of growth of the epithelium in these tumors is varied. The most characteristic pattern is one in which there are many islands of epithelium bordered by regimented columnar cells. In the center of these islands is a loose network of epithelial cells resembling the stellate reticulum of a developing tooth. Such epithelial units are highly suggestive of distorted enamel organs (Fig. 1). In some tumors the epithelial islands are packed together, in others there is a moderate amount of connective tissue between them. Occasionally the epithelial cells will grow in double rows forming strands of epithelium which simulate extensions of the dental lamina (Fig. 2). At times the histologic pattern of an ameloblastoma may be such that difficulty is encountered in identifying the tumor as one which has arisen from odontogenic epithelium. Usually, however, careful searching will reveal epithelial structures which simulate those seen in some early stage of tooth development.

Most ameloblastomas eventually become cystic. The cystic change may only be apparent microscopically in some tumors, but in others the cysts are large enough to be identified grossly (Fig. 3). Cysts form when islands of neoplastic epithelial cells become so large that the central cells die and liquefy because their source of nutrition (stromal capillaries) is so distant. As the cysts enlarge the cortical plates may bulge, producing a deformity. When taking a biopsy specimen from a markedly cystic tumor, it is important to get representative material. If only a cyst is procured for histologic examination, the diagnosis may be missed for one of these cysts may look like any other cyst which is lined by stratified squamous epithelium.

Ameloblastomas are sometimes described as being primitive, plexiform, stellate, follicular, acanthomatous or adenomatous. These adjectives when used in conjunction with the term ameloblastoma do not imply specific sub-varieties of the tumor, but only refer to the predominant histologic pattern. They are of academic interest only and do not alter the basic diagnosis of ameloblastoma. The descriptive significance of each is as follows: *primitive*—structures resembling extensions of the dental lamina (Fig. 2); *plexiform*—anastomosing cords and islands of epithelium (Fig. 4); *stellate*—epithelial cells simulating the stellate reticulum; *follicular*—epithelial islands mimicking enamel organs (Fig. 1); *acanthomatous*—squamous metaplasia of the stellate reticulum in many of the enamel organ-like structures; *adenomatous*—many duct-like formations. Some of the tumors having the latter pattern are so distinctive and consistent in their
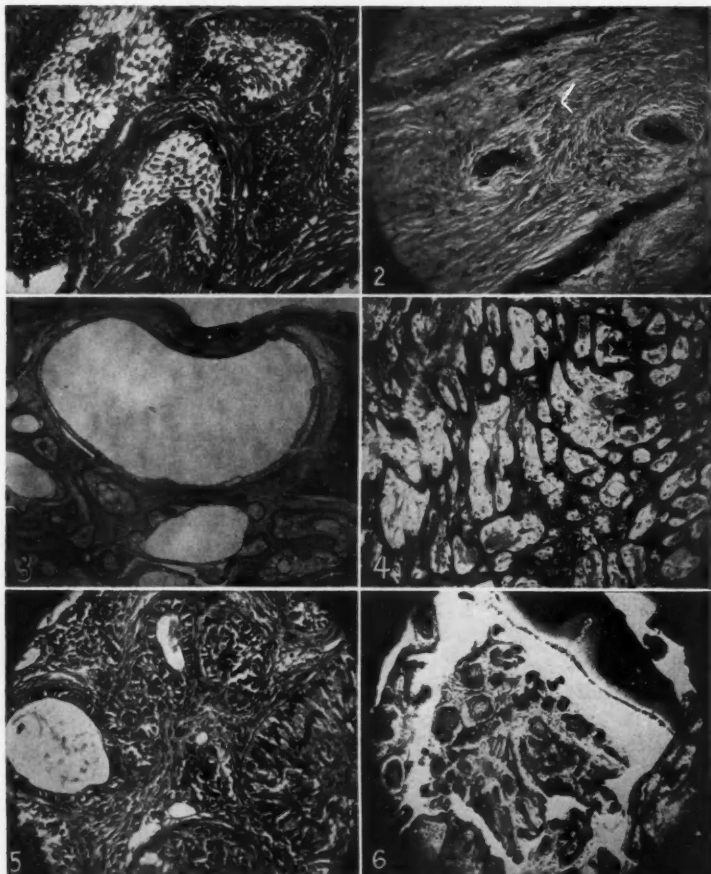
Fig. 1. Ameloblastoma, follicular pattern. Three epithelial units mimicking enamel organs are evident.

Fig. 2. Ameloblastoma. Near top and bottom of the photomicrograph are long narrow epithelial structures similar to extensions of the dental lamina seen in normal tooth development.

Fig. 3. Ameloblastoma. Large cyst is apparent at top of the picture. Elsewhere epithelial units are in various stages of cystic degeneration.

Fig. 4. Ameloblastoma, plexiform pattern.

Fig. 5. Adenoameloblastoma. A large duct is seen at left and a smaller one is apparent near the top border. At far right, ameloblast-like cells appear to have secreted an amorphous material.

Fig. 6. Odontoameloblastoma. Dentin and enamel are seen near upper right hand corner. Neoplastic epithelial cells arranged mainly in a follicular pattern are apparent in the remainder of the picture. (Courtesy of Dr. Donald A. Kerr.)

appearance that they have been classified by some as a specific variant of the ameloblastoma, i.e., *adenoameloblastoma* (Fig. 5). This tumor consists of small and medium-sized, clear spaces, each bordered by a single row of broad columnar cells. Adjacent to the ductile structures are double rows of tall, narrow columnar cells with basally located nuclei. Within the strands formed by the double row of cells there is usually amorphous material which stains blue with hematoxylin and eosin.

The *hemangioameloblastoma* as a specific entity is much in dispute. It is purported to be a compound tumor consisting of a hemangioma in association with an ameloblastoma. The few cases that have been reported do show many relatively large endothelium-lined spaces packed with red blood cells, but whether or not these vascular spaces should be interpreted as representing a hemangioma is controversial. There is no argument about the ameloblastomatous portion of the lesion. This so-called entity may be nothing more than a well vascularized ameloblastoma.

The *odontoameloblastoma* is an ameloblastoma which is associated with an odontoma (Fig. 6). The odontomatous portion of the lesion is composed of calcified dental tissues which are either haphazardly arranged or form structures resembling small teeth. This part of the tumor, being an anomaly and not a neoplasm, is relatively unimportant from the standpoint of the patient's welfare. The neoplastic portion is no different from any other ameloblastoma and its component epithelial cells may grow in such a manner as to form any of the histologic patterns previously described.

Generally, ameloblastomas are slow-growing tumors. Some show marked local aggressiveness. A few have been reported as being malignant, but authenticated cases of true metastasis are extremely rare. The presence of ameloblastoma in the lung is not conclusive evidence of metastasis, for neoplastic cells may have been aspirated and implanted in this organ. The ameloblastoma is primarily a central tumor of bone but will at times erode the cortical plates and extend into the soft tissue, especially after incomplete surgical interference. These tumors are usually not encapsulated and thus cannot be easily enucleated. Simple curettement seldom effects a cure.

Ameloblastomas are seen much more frequently in the mandible than in the maxilla. The most common site is the angle of the lower jaw. These tumors are most often discovered during the fourth decade of life. Frequency of occurrence is not related to sex or race.

Ameloblastomas destroy bone and so they are recorded roentgenographically as radiolucent areas. Their appearance in a roentgenogram may be similar to that of many other lesions which cause a loss

of bone. Generally, however, the area of decreased density is traversed by many curved, radiopaque lines which seem to divide the abnormal area into chambers of various sizes (Fig. 7). When these locules are small and of nearly the same size the pattern formed resembles a honeycomb (Fig. 8). Sometimes an ameloblastoma is of unilocular character and occasionally it may appear as a small nonde-
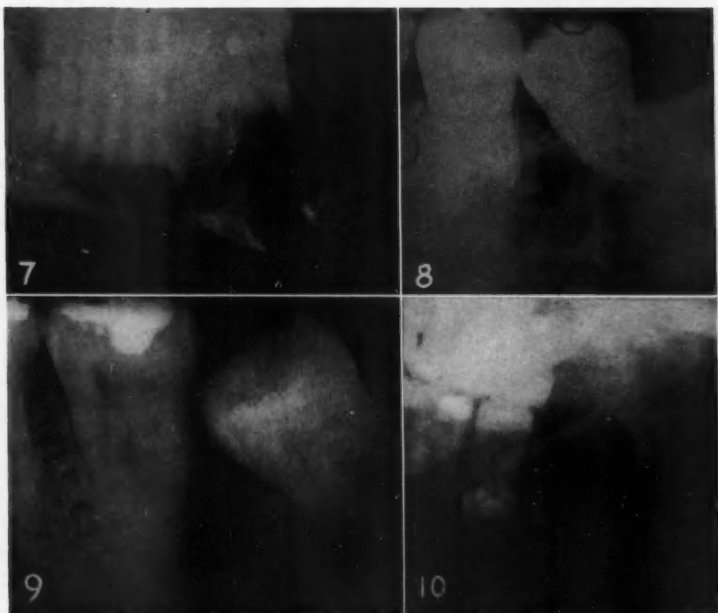
Fig. 7. Lateral jaw roentgenogram showing multicystic appearance of an ameloblastoma.

Fig. 8. Periapical roentgenogram of an ameloblastoma. Note honeycomb appearance of the cyst-like areas.

Fig. 9. Small area of radiolucency reflecting the bone loss caused by an ameloblastoma. An example of how innocuous an early lesion may appear roentgenographically.

Fig. 10. Roentgenogram of a dentigerous cyst. Ameloblastoma was found in the wall of this cyst when it was examined histologically.

script area of radiolucency (Fig. 9). In the instance of an odontoameloblastoma, radiopaque structures are seen in the area of decreased density. Some ameloblastomas masquerade as dentigerous cysts roentgenographically (Fig. 10). When such lesions are operated, the area which was thought to be a cyst in the roentgenogram may be filled with tumor tissue. In other instances a cyst may be present but ameloblastoma is found in its wall.

The diagnosis of ameloblastoma should never be based on roentgenographic evidence alone. The roentgenographic as well as the clinical findings are helpful in making the diagnosis but the burden of proof must lie primarily with the histologic examination.

Odontogenic Fibroma

The odontogenic fibroma arises from the mesodermal components of the tooth germ, specifically from the dental papilla or from the dental sac. Histologically it is composed of many stellate fibroblasts and delicate interlacing fibers (Fig. 11). It almost duplicates the appearance of dental pulp in a young person. Generally, dense fibrous tissue is not observed in this tumor. Odontogenic epithelium may be seen in association with the lesion and, in fact, its presence is necessary if one is to be certain as to histogenesis of the neoplasm. The epithelium is quiescent and is not part of the tumor. Odontogenic fibromas appear roentgenographically as either unilocular or multilocular areas of radiolucency.

Fibroameloblastoma

This neoplasm arises from both the mesodermal and ectodermal constituents of the tooth germ. It is therefore a mixed tumor which is composed of neoplastic connective tissue and neoplastic epithelial tissue. The epithelial portion grows to form the various structures seen in the ordinary ameloblastoma. The connective tissue component may be rather delicate (Fig. 12) or it may be relatively dense (Fig. 13). The fibroblasts in some cases vary greatly in size, shape and staining reaction.

Histologically, the fibroameloblastoma looks much like the ameloblastoma except that in the former the connective tissue shows evidence of neoplastic proliferation and is out of proportion to the amount that would be necessary to support the epithelial growth.

The roentgenographic findings, clinical features and growth potential of the fibroameloblastoma are similar to those of the ameloblastoma.

BLASTOMATOID LESIONS

The blastomatoid lesions (pseudo-neoplasms) of odontogenic origin include only those entities arising from tooth-forming tissues which demonstrate a fair degree of growth but do reach a stage when enlargement ceases. They thus differ from neoplasms, which have the potentiality for unlimited growth. The only odontogenic lesions which
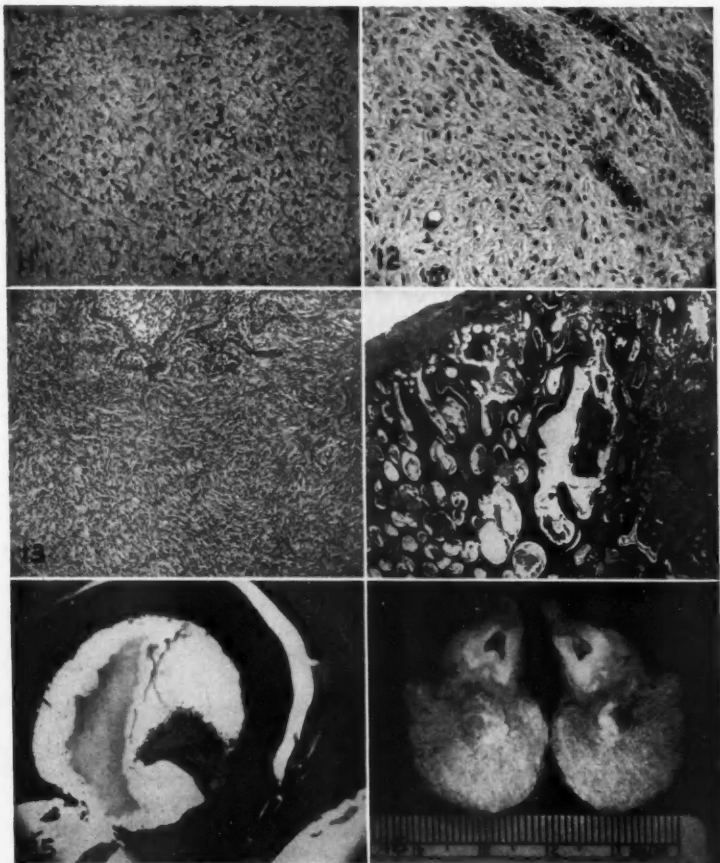
Fig. 11. Odontogenic fibroma. The neoplastic connective tissue is of delicate character comparable to that seen in the dental pulp.

Fig. 12. Fibroameloblastoma. Both the epithelium (upper right) and the connective tissue are neoplastic. Collagen is not present and the connective tissue is loosely arranged.

Fig. 13. Fibroameloblastoma. The neoplastic fibrous tissue is relatively dense and quite cellular. Units of neoplastic epithelial cells are seen near the top border.

Fig. 14. Low power view of a complex composite odontoma. The darker areas are dentin; the clear spaces formerly contained enamel. The capsule is seen at the upper left.

Fig. 15. High magnification of the odontoma seen in Figure 14. The circular clear space contained enamel prior to specimen preparation. A remnant of enamel matrix is seen at 4 o'clock. Dentinal tubules are apparent at right and near bottom margin.

Fig. 16. Complex odontoma attached to a molar tooth. The specimen was sawed in half before decalcification. The white areas seen on the cut surface are enamel.

meet the above criteria are the *composite (mixed) odontomas* and the *cementomas*.

Dens in dente, geminated teeth, fused teeth and enamel pearls are sometimes classified as odontogenic tumors, but it does not seem proper to consider them as tumors even in the widest meaning of the term.

Composite (Mixed) Odontoma

Composite (mixed) odontomas are tumorous anomalies composed mainly of calcified dental tissues. The words "composite" and "mixed" signify that these entities contain tissues derived from both ecto- dermal and mesodermal portions of the tooth germ. Composite odon-

Fig. 17. Periapical roentgenogram of a complex odontoma. A radiolucent line (the capsule) is seen at left.

tomas therefore contain enamel (from ectoderm) and one or more of the mesodermal derivatives, i.e., dentin, cementum or pulp.

Two forms of the composite (mixed) odontoma are recognized: (1) the complex type and (2) the compound type.

The *complex composite odontoma* consists of a single mass of dental tissues haphazardly arranged. Usually the predominate tissue is den- tin. Throughout the dentin, clear spaces are generally seen (Fig. 14). These spaces were occupied by enamel before decalcification of the specimen in the laboratory. Much of the dentin is very irregular but usually a few tubules may be identified (Fig. 15). Most complex odontomas are surrounded by a connective tissue capsule. Some are fused to fully formed unerupted teeth.

Grossly, complex odontomas are of a light yellow color because they are predominantly of dentin. The cut surface of an odontoma, bisected prior to decalcification, will reveal small spherical pearl-colored areas of enamel scattered throughout the yellow dentin (Fig. 16).

Roentgenographically, complex odontomas appear as single spheroidal radiopacities of varying sizes (Fig. 17). The fibrous capsule is recorded in the roentgenogram as a fine, dark line.

A *compound composite odontoma* consists of the same tissues that are seen in the complex type but they are arranged to form structures which are more or less recognizable as teeth. The components of a compound odontoma are usually described as denticles. Compound odontomas may contain only 2, 3 or 4 denticles or as many as 200 such structures. Some of the denticles may duplicate specific tooth forms while others may be tiny, irregular, seed-like particles. Around each denticle there is a band of fibrous tissue (Fig. 18).

Fig. 18. A portion of a compound composite odontoma. The denticles are separated by fibrous tissue.

Fig. 19. Periapical view of a small compound odontoma. Four denticles can be identified.

The roentgenographic picture of a compound odontoma is very characteristic (Fig. 19). In it may be seen discrete objects which have the shape and density of teeth. Each denticle and the whole tumor are surrounded by radiolucent lines.

Cementoma

The term "cementoma" is usually reserved for those cases of apical hypercementosis which are preceded by periapical bone resorption. It is generally recognized that there are at least two stages in the formation of a cementoma. First, periapical bone is destroyed and re-

placed by fibrous tissue. Later the fibrous tissue is gradually replaced by cementum. Several years may elapse between the first and second stage. The cause of this phenomenon is not known.

Cementomas do not affect tooth vitality. They may be associated with any tooth but most frequently with the mandibular incisors. These overgrowths do not usually exceed 2 cm. in diameter, but often involve more than one tooth. Generally, there are no symptoms connected with the cementoma, and the presence of such an entity is no indication for extraction.

During the first stage of a cementoma the lesion is recorded roentgenographically as a spherical radiolucency (Fig. 20), quite similar
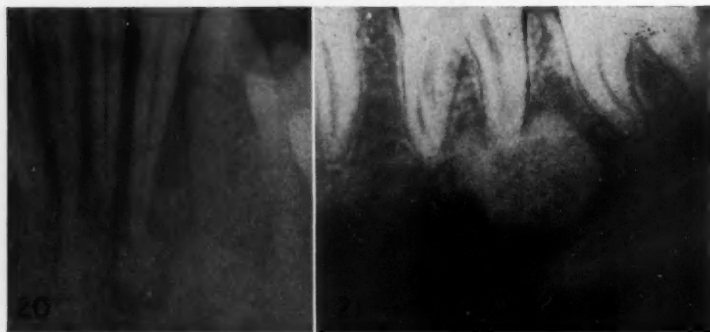
Fig. 20. Cementomas. A small radiolucent area (the first stage of a cementoma) is seen about the apex of the central incisor. Radiopaque material has partially replaced the fibrous tissue at the apex of the lateral incisor.

Fig. 21. Fully developed cementoma associated with a molar tooth. A thin radiolucent line surrounds the cemental mass.

to that commonly associated with periapical inflammation. A positive vitality test would eliminate the latter diagnosis. As the second stage begins, some degree of increased density becomes apparent in the radiolucent area. When the second stage is completed the roentgenogram reveals a definite radiopaque area bordered usually by a dark line (Fig. 21).

SUMMARY

Odontogenic tumors arise within the jawbones from the tooth-forming apparatus. Some are true neoplasms and others, which do not have the potentiality for unlimited growth, are termed blastomatoid lesions. The odontogenic neoplasms are the ameloblastoma, the odontogenic fibroma and the fibroameloblastoma. The blastomatoid lesions are the composite odontoma and the cementoma.

The neoplasms are primarily benign lesions, but may be very destructive locally. Roentgenographically, they usually appear as unilocular or multilocular areas of radiolucency, but biopsy is necessary to establish a diagnosis.

The blastomatoid lesions are limited in their growth. The composite odontomas are composed of calcified dental tissues derived from both ectoderm and mesoderm. The compound type consists of rudimentary teeth; the complex type is a single mass of irregularly arranged dental tissues. The cementoma develops slowly as a fibrous mass at the periapex, which gradually is replaced by cementum.

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Primary and Secondary Malignant Tumors
of the Jawbones

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Primary malignant tumors of bone are fortunately rare and for any given tumor the jaws are among the bones less frequently involved. The rarity of these lesions makes it difficult for any one clinician or even one institution to develop a wide experience with them. Coley[4] stated that only 35 confirmed cases of sarcoma of the jaw bones were seen over an 18 year period at Memorial Hospital in New York City. Barnes Hospital in St. Louis has very active orthopedic and head and neck services with many referred cases. About 25 cases of malignant bone tumors of all regions are seen there in a year. Table 1 shows the distribution and frequency of the various types of malignant bone tumors over a 5 year period.

TABLE 1. *Malignant Bone Tumors in Jawbones and Elsewhere, 5 Year Period*

	NO. OF CASES IN BONES OTHER THAN THE JAWS	NO. OF CASES IN MAXILLA OR MANDIBLE
Osteosarcoma	41	3
Chondrosarcoma	30	1
Fibrosarcoma	12	1
Ewing's sarcoma	12	0
Reticulum cell sarcoma	8	0
Myeloma	24	0

In spite of their infrequent occurrence, sarcomas of the jaws are of importance to the dentist from the standpoint of diagnosis and differential diagnosis. Diagnosis of many of these tumors is very difficult, as the early signs and symptoms are often confused with infec-

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tions of dental origin or more common benign cysts and tumors involving the jaws.

The classification of primary bone tumors is complex and still controversial, partly because of the wide range of microscopic patterns present in some of these tumors and the difficulty in determining the cell of origin of certain types. In this article primary bone tumors will be discussed under the following headings: osteosarcoma, chondrosarcoma, fibrosarcoma, Ewing's sarcoma, reticulum cell sarcoma, and plasma cell myeloma.

OSTEOSARCOMA

Osteosarcoma (osteogenic sarcoma) is the most common primary malignant tumor of bone. This tumor arises from bone-forming mesenchymal tissue in the medullary cavity of a bone. Osteosarcomas show a wide spectrum of changes ranging from well differentiated tumors which produce considerable new bone to highly anaplastic tumors in which bone formation is scant. Osteosarcomas are usually found in patients between 10 and 25 years of age, although occasional cases may be seen in all age groups. Most instances in older persons occur in those with Paget's disease (osteitis deformans). About one-third of all osteosarcomas develop in the region of the knee, i.e., the lower portion of the femur and upper portion of the tibia. The upper portion of the humerus is also a common site. Osteosarcomas of the oral region are most often found in the mandible.

Signs and Symptoms. Pain is often an early symptom of osteosarcoma of the jaws. This pain may at first be sporadic but later in the course of the disease it becomes more constant. Swelling of the jaws, loosening of the teeth and paresthesia are also important symptoms. Growth of the tumor is often quite rapid and invasion of the soft tissues about the bone leads to the development of a large tumor mass. In most reported series, the present of a mass was the most frequent complaint causing the patient to seek professional care. Sherman[6] reports that 14 of 17 patients with osteosarcomas of the mandible consulted dentists either originally or very early in the course of the disease.

The roentgenographic features vary with the extent of the bone produced by the tumor. In the sclerosing form, large amounts of dense, structureless bone are formed. Cortical destruction, periosteal reaction and development of a soft tissue mass containing radiating spicules of tumor bone are characteristic (Fig. 1). The lytic forms of osteosarcomas are characterized by little evidence of bone formation and show only bone destruction with extension through the cortex

and a soft tissue mass. In other cases the roentgenogram shows a combination of bone destruction and bone formation.

Diagnosis, Treatment and Prognosis. Adequate biopsy examination is essential for diagnosis but the microscopic findings must be correlated with the roentgenographic and clinical features. Errors have been made in both over- and under-diagnosis of this tumor. Radical resection of the involved bone is the only treatment. Radiation therapy is seldom effective. In spite of prompt, radical treatment, the outlook for survival with osteosarcoma is very poor. The 5 year survival rate is only about 5 per cent. Unfortunately, in many cases the tumor

Fig. 1. Roentgenogram of a surgical specimen of an osteosarcoma of the mandible, demonstrating a large extra-osseous mass which contains tumor bone.

has already metastasized to the lungs before the diagnosis is established. Older reports indicating a higher cure rate usually included less malignant tumors such as chondrosarcomas and fibrosarcoma under the general heading of osteogenic sarcoma. Some of the older "cured" cases of osteosarcoma have proved on re-examination to be examples of benign processes such as fibrous dysplasia or ossifying fibromas.

CHONDROSARCOMA

Chondrosarcomas are neoplasms composed of malignant cartilage cells. These tumors may arise from pre-existing benign cartilagenous tumors (osteochondromas or enchondromas) or from bone which was previously not abnormal. Although earlier classifications grouped

chondrosarcomas under the general heading of osteogenic sarcomas, most authorities today consider chondrosarcomas as a separate entity. This separation is justified on the basis of age incidence, behavior and prognosis. Chondrosarcomas are not as common as osteosarcomas and tend to occur in an older age group. Growth is slower and there is less likelihood of metastasis. The prognosis for a patient with chondrosarcoma is generally better than for the patient with osteosarcoma.

In view of the paucity of reported cases, chondromatous tumors of the jaws must be considered as rare neoplasms. They appear to occur somewhat more frequently in the anterior maxillary region but have been reported in all areas of the jaws. Although both benign
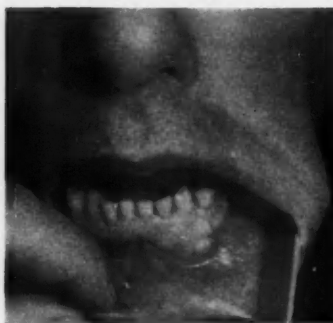
Fig. 2.   Fig. 3.

Fig. 2. A chondrosarcoma presenting as a painless, irregular enlargement of the aveolar process in a 35 year old man.

Fig. 3. Roentgenogram of this case showing an area of irregular mottled calcification. The root of the first premolar is resorbed.

forms (chondromas) and malignant types (chondrosarcomas) have been described in the jaws, the experience of many oral pathologists indicates that all cartilagenous tumors of the jaws must be regarded with suspicion.

Signs and Symptoms. Chondromatous tumors of the jaws are frequently peripheral in type, tending to grow out from the surface of the bone (Fig. 2). A painless, localized swelling of the alveolar process, maxilla or mandible is the most common presenting symptom. Unlike osteosarcomas, pain, loosening of teeth or paresthesia appear to be uncommon symptoms. Roentgenographic interpretation of these tumors is difficult. The involved bone tends to show a spotty or irregular calcification which alternates with irregular areas of radiolucency. The roots of the teeth in the area may show irregular resorption (Fig. 3). The extent of involvement is often greater than the roentgeno-

graphic appearance indicates. In some cases, at surgery the bone has been found to be extensively invaded although the roentgenograms were interpreted as normal.

Diagnosis, Treatment and Prognosis. Diagnosis can be made only by histologic examination. The separation of benign from malignant cartilagenous tumors on histologic grounds may prove difficult in some cases and the pathologist may tend to under-diagnose this tumor. In some cases the diagnosis of chondrosarcoma rather than chondroma has been forced by the clinical course rather than the histologic features. It is probably best to consider all cartilagenous tumors of the jaws as at least potentially malignant. Treatment consists of wide surgical excision. If this is not done the tumor tends to recur promptly. Even with frankly malignant chondrosarcomas, metastasis tends to be relatively slow and late in the course of the disease. The probability of cure is greater than with most forms of bone sarcoma if the diagnosis is made early and prompt radical surgery is performed. Metastasis to the lungs is the chief hazard. In a few cases chondrosarcomas may be highly malignant tumors from the onset and run a clinical course similar to osteosarcomas.

FIBROSARCOMA

Fibrosarcoma of bone is a malignant tumor of fibroblastic cells. These tumors are differentiated from osteosarcomas and chondrosarcomas in that no bone, osteoid tissue or cartilage is produced by the tumor cells. Central fibrosarcomas originate within the medullary cavity of a bone while the periosteal type originates in the covering soft tissues and may invade the bone by direct continuity. In advanced cases it is often difficult to determine if a tumor began centrally or periosteally.

Signs and Symptoms. Fibrosarcomas have been observed in both mandible and maxilla and occur over a rather wide age range. Recent reports indicate that this tumor may arise in areas previously subjected to heavy radiation therapy. Periosteal fibrosarcomas of the jaws are characterized by a slow or rapid development of a soft tissue mass on the surface of the bone. Roentgenographic examination may reveal irregular destruction of the bone cortex. Central fibrosarcomas develop insidiously and may be first associated with pain or the loosening of teeth. The roentgenographic features are not distinctive and show a moth-eaten or mottled rarefaction of the affected bone (Fig. 4). These findings may closely simulate an inflammatory process, which may delay the diagnosis. As the tumor enlarges it may destroy the cortex and form a large soft tissue mass around the bone.

Diagnosis, Treatment and Prognosis. Diagnosis can be made with certainty only by microscopic examination of the tumor. Wide surgical excision is the only effective treatment. Fibrosarcomas show a wide range of activity. Some are well differentiated, grow slowly and offer

Fig. 4. Roentgenogram of a central fibrosarcoma of the mandible in a girl, age 12. The initial symptom was a sudden loosening of her second molar. This was extracted and a mass of abnormal tissue grew out of the socket. Biopsy showed a fibrosarcoma. The roentgenogram reveals an irregular destructive process in the molar region with loss of bone distal to the first molar. Radical resection of the mandible was performed and the patient survived 3 years before fatal lung metastases developed.

a good prognosis. In other cases, however, the tumor may be highly anaplastic and very malignant. Metastasis to the lungs is the chief hazard.

EWING'S SARCOMA

This tumor is one of the most controversial lesions in bone pathology and some authorities even doubt its existence as an entity. It presumably arises from the young reticular cells of the bone marrow and is composed of small round or oval cells with little or no visible cytoplasm. The tumor cells do not form reticulum fibers, osteoid tissue or bone. Most cases are observed in persons under the age of 25 years. Pain is a common initial symptom. The tumor produces osteolytic changes in the medullary cavity of the affected bone, and thickening of the cortex with periosteal new bone proliferation is characteristic.

A few cases of Ewing's sarcoma of the mandible have been reported. Pain of the jaws appears to be the most common symptom. The roentgenographic appearance is not specific and shows only irregular osteolytic changes. Biopsy examination will show a small round cell neoplasm. Diagnosis of Ewing's sarcoma is difficult to substantiate by microscopic examination, as lymphosarcoma, metastastic carcinoma and neuroblastoma must be excluded. These neoplasms

may produce similar clinical, roentgenographic and histologic findings. The prognosis of Ewing's sarcoma is very bad. The tumor is radiosensitive but seldom radiocurable. The tumor often spreads to other bones and particularly to the lungs.

RETICULUM CELL SARCOMA

Primary reticulum cell sarcoma of bone has only been recognized as an entity since 1939. This tumor is derived from reticulum cells of the marrow of the affected bone and its cell type is identical with the reticulum cell sarcomas of lymph nodes and other blood-forming tissues. Most cases occur in the long bones of middle-aged persons. The tumor is frequently painful and causes a large destructive lesion within the affected bone. In the long bones there is a marked tendency toward pathologic fracture.

Gerry and Williams[5] reported 2 cases involving the mandible and found 6 previously reported cases in the literature. Pain which may or may not be associated with a palpable swelling of the jaw is the most common initial symptom. The roentgenographic appearance is characterized by an irregular area of bone destruction, but it is not diagnostic, however, and may be confused with an inflammatory process. Loosening of the teeth and destruction of the lamina dura may be noted when the tumor involves a tooth-bearing area of the jaw. Diagnosis is possible only by microscopic examination of the tumor tissue. Surgery and radiation therapy have been advocated for treatment. With adequate therapy the survival rates appear more favorable than with many forms of bone cancer. Cure rates as high as 50 per cent have been reported.

PLASMA CELL MYELOMA

Plasma cell myeloma is a malignant process which usually arises in abnormal plasma cells of the bone marrow, causing extensive bone destruction and progressing to a fatal termination. Most cases are seen in patients over 50 years of age, and men are affected about twice as often as women. The disease shows a wide variation in its course. It is most often observed as multiple or diffuse involvement of a number of bones at the time of initial diagnosis. A number of cases of apparently solitary myelomas of bone have also been reported. The nature of these lesions is controversial. Many feel that it is unwise to regard any of these "solitary" lesions as benign, as the process may terminate in diffuse myeloma many years after the diagnosis of a "solitary" lesion. The extramedullary plasma cell tumor, which is most frequently seen in the soft tissues of the oral cavity and upper

air passages, presents a similar problem. These tumors are unpredictable in that they may remain localized for long periods and then terminate in diffuse myeloma or expand rapidly into an invading tumor that destroys bone and adjacent tissue.[3]

Multiple myeloma most frequently involves the vertebral column, ribs, skull, pelvis and femur. Bone pain, most often in the back, is a frequent initial symptom. In a high percentage of patients with myeloma there will be an elevation of their serum globulins owing to the presence of abnormal globulins, chiefly of the gamma fraction. An abnormal protein, the Bence-Jones protein, may also be detected in the urine.

Involvement of the jawbones by myeloma is not infrequent and probably occurs more often than is generally recognized. The mandible, particularly the molar regions, is affected more often than the maxilla. In some cases the initial signs and symptoms of myeloma may appear in the oral cavity. Bruce and Royer[1] reviewed 59 cases of multiple myeloma from the Mayo Clinic. Seventeen of these patients exhibited evidence of myeloma in the jaws. In 7 of these cases the initial symptoms were first observed in the jaws.

Signs and Symptoms. Pain, swelling, expansion of the jaws, numbness and mobility of teeth are common symptoms of myeloma involving the mandible or maxilla. In some cases perforation of the tumor through the bone cortex may result in the appearance of an epulide lesion on the gingiva. Myeloma of the jaws often appears roentgenographically as relatively small, multiple rounded areas of radiolucency with little circumferential osteosclerotic reaction (Figs. 5 and 6). This appearance cannot be considered diagnostic in itself and may be produced by metastatic carcinomas of the breast or thyroid, reticulum cell sarcomas and some cases of hyperparathyroidism. In many cases myelomas may not show this classic roentgenographic appearance. The lesion may present as a large, solitary area of bone destruction which may be associated with the roots of one or several teeth and closely simulate a large dental granuloma or periodontal cyst. In other instances a myeloma of the jaws may appear roentgenographically as a "cystic," trabeculated, honeycombed or "soap bubble" type of lesion which closely resembles the appearance of giant cell lesions or ameloblastomas. This appearance is especially common in the "solitary" myelomas.

Diagnosis, Treatment and Prognosis. The presence of multiple, sharply circumscribed radiolucent defects in the jaws, particularly in an older patient, should raise a suspicion of myeloma. In other cases the nature of the disease may only be detected after surgical intervention and microscopic examination of tissue removed from an osteo-
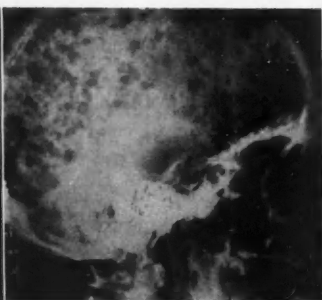
Fig. 5.   Fig. 6.

Fig. 5. Roentgenogram showing a sharply circumscribed radiolucent area in the mandible of a woman, age 60. Several less well defined areas are also present in the ascending ramus. (Courtesy of Westfield State Sanatorium, Westfield, Mass.)

Fig. 6. Lateral skull film of the same patient showing multiple osteolytic defects. (Courtesy of Westfield State Sanatorium, Westfield, Mass.)

lytic defect in the jaw or after excision and microscopic examination of an epulide lesion. Final diagnosis usually requires skeletal roentgenograms, bone marrow aspiration and laboratory examinations of the serum and urine. Multiple myeloma is invariably fatal although life may be prolonged by radiation therapy or use of urethane.

METASTATIC CANCER INVOLVING THE JAWS

Metastatic carcinoma is the most common form of cancer involving bone. The actual incidence of metastases to the skeleton is difficult to ascertain but is undoubtedly higher than is generally realized. Careful autopsy studies have shown that over two-thirds of breast carcinomas, one-half of prostate carcinomas, one-third of lung carcinomas and one-fourth of kidney carcinomas will spread to one or more bones before death. Although metastatic lesions may on occasion be observed in any bone, the vertebrae, ribs, pelvis, femur and skull are the most frequently involved.

Clinically evident metastasis of cancer from distant sites to the jaws is not common. Metastasis in this sense should be distinguished from direct invasion of the jawbones by primary oral or salivary gland carcinoma, which occurs frequently. Unrecognized metastatic involvement of the jaws is probably more common than is indicated by the literature. In most instances osseous metastasis occurs relatively late in the course of the disease and roentgenographic or necropsy studies of the oral regions are seldom made in such cases. Castigliano and Rominger[2] reviewed 176 cases of metastatic malignancy involving the jaws reported between 1902 and 1953. The primary tumors most fre-

quently accounting for jaw metastasis were carcinomas of the thyroid, kidney, breast, lung and prostate. Metastasis to the jaws by carcinomas of the large intestine, ovary and testes have also been reported. The mandibular posterior area and the region of the angle of the mandible is the area most frequently involved by metastatic tumors.

Signs and Symptoms. Metastatic involvement of the jaws may cause a wide variety of symptoms. The patient may complain of pain, swelling, toothache, loosening of teeth, numbness of the lip or a soft tissue mass. In two personally observed cases the patients' initial complaints were that a previously satisfactory lower denture had suddenly become ill fitting. In both cases roentgenographic examinations showed

Fig. 7. Roentgenogram of a metastatic carcinoma of the lung presenting as a "cystic" lesion in the mandible. This lesion was the first evidence of the patient's malignant disease.

destruction of bone with expansion of the mandible and partial obliteration of the buccal sulcus.

Most carcinomatous metastases to bone cause destructive (osteolytic) lesions. The roentgenographic picture may vary from a well-circumscribed radiolucent area resembling a "cyst" to a diffuse moth-eaten appearance which may simulate an inflammatory process or a primary bone tumor (Fig. 7). Involvement of the alveolar process about the roots of teeth may on occasion present the clinical and roentgenographic features of periodontal disease. Some forms of metastatic carcinoma, particularly those originating in the prostate gland, cause proliferation of new bone at the site of metastasis. These areas will appear roentgenographically as multiple irregular areas of increased density. In some instances both osteoclastic and osteoblastic metastases may be present in one bone.

Diagnosis. The roentgenographic features of metastatic carcinoma involving the jaws can seldom be considered diagnostic as they may

simulate primary cancer, benign cysts or tumors or infections of dental origin. Consideration of the possibility that any given lesion may represent metastatic malignancy is an important initial step in the diagnosis. A complete and accurate history of previous illnesses or operations is also essential. In most cases, however, final diagnosis rests upon microscopic examination of tissue removed from the lesion in question. Of particular interest and importance to the dentist are those cases in which a metastatic lesion in the jaws is the first symptom of malignant disease. These patients who have an undetected primary carcinoma in some other area of the body may consult a dentist for symptoms referable to a metastatic lesion in the jaw. In such cases biopsy examination is the only means of diagnosis.

The prognosis for the patient with bone metastasis is usually very grave. However, surgical excisions of solitary metastatic foci after control of the primary tumor are being performed more frequently and some apparent cures have been obtained.

Sarcomas from distant sites may on rare occasions also metastasize to the jaws.

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The Treatment of Oral Cancer

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Cancer of the mouth is treated by the same methods as cancer in other anatomic areas. Surgery was the first effective method of treatment and many descriptions of radical surgery can be found in old textbooks. These procedures were abandoned years ago because of sepsis, but with the advent of the antibiotics many of them have been found to be practical and effective. When radiation became popular in the early decades of this century, most oral cancer was treated by this method. However, following the initial enthusiasm, it was found that there was morbidity associated with adequate radiation, and that many tumors were not controlled. The recent refinements in x-ray and the development of radioactive isotopes have increased the usefulness of this type of treatment for oral cancer.

The choice of treatment, whether radiative or surgical, is dependent on location, histologic type, previous treatment and whether the lesion is primary or metastatic.

Surgical accessibility is necessary for adequate removal of the tumor. Complete resection is more feasible if the location is in the anterior or middle portion of the oral cavity than if in the posterior region or the pharynx. Many tumors located posteriorly are anaplastic and respond favorably to radiation therapy. If the involved tissue is proximate to bone, the underlying bone will receive much of the radiation and therefore surgical resection is the preferred treatment.

Squamous carcinoma is the most common histologic type occurring in the oral cavity. Adenocarcinomas of the salivary gland and malignant melanomas occasionally appear. Radiation can completely control squamous carcinoma in many instances. (Generally the response to radiation parallels the degree of anaplasia.) Salivary gland adenocarcinomas and malignant melanomas are radioresistant and should be treated by wide surgical resection.

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The previous treatment of a lesion definitely affects the method of therapy to be employed. If an area has been irradiated with curative intent and the tumor has persisted or recurred, radiation should not be used again unless palliation only is expected. Radiation effect in tissue is permanent. On the other hand, when a lesion has been previously excised and there is recurrence, one may still choose between further surgery or radiation.

Most authorities agree that the treatment of choice for metastatic lesions in the neck is surgical resection. Radiation is used when necessary as a supplement to surgery.

PRINCIPLES OF SURGICAL MANAGEMENT

The following principles should be considered in the surgical management of oral cancer.

Adequate Resection. The first attempt to resect a tumor offers the best opportunity for control. In the head and neck area where important anatomic structures are proximate, there is often hesitation in sacrificing enough tissue. Resection of adequate tissue around a malignant tumor is imperative regardless of what must be sacrificed.

Cosmetic Appearance. Patients naturally do not wish unnecessary mutilation, especially about the face. However, one is often faced with the necessity of mutilation to produce a cure. The surgeon offering this treatment has to be convinced of its necessity before he can be assuring to his patient. Cosmetic reconstruction can be performed after the lesion is definitely under control.

Tracheostomy. Edema, impairing the airway, often develops following extensive operative procedures about the mouth and neck. To eliminate this hazard, tracheostomy is routinely performed as a prophylactic measure at the termination of such operations. Morbidity is low, the lifesaving potential great. A tracheostomy tube is allowed to remain until it may be "corked" continuously for 48 hours.

Infection Control. The oral cavity contains many organisms which are pathogenic if they contaminate the deeper tissue planes. Prior to the antibiotics, sepsis was commonplace in surgical procedures involving the oral, pharyngeal or laryngeal cavities. Now it is customary to administer antibiotic therapy routinely until danger from infection has passed. In addition, oral irrigations with dilute hydrogen peroxide are helpful in controlling sepsis.

Tube Feedings. If the incision involves the oral cavity, feedings by mouth should not be permitted during the healing period. Tube feedings offer three distinct advantages: (1) direct contamination of the

wound is prevented; (2) continual chewing and swallowing of food, placing stress on the suture line, is avoided; and (3) adequate nutrition is maintained during the healing period.

PRINCIPLES OF RADIATION THERAPY

The following principles are to be observed when treating an oral lesion by radiation therapy.

Adequacy. The amount of radiation delivered to the tumor should be adequately high on the initial treatment of the lesion. Radiation failures are often the result of sublethal total dosage or misdirected beams. The physician must know whether he is seeking curative or palliative results and treat accordingly.

Dental Damage. Heavy radiation is damaging to the teeth as well as the jaws. Obliterating endarteritis is initiated which renders the bone more susceptible to infection. Even though the patient escapes apparent damage at the time of treatment, he may encounter serious disability years later when dental treatment is necessary. Heavy antibiotic therapy should be given in these situations.

The teeth and jawbone should be shielded when treating intraoral malignant tumors; if this is not possible, the teeth should be extracted prior to radiation. In fact, some authorities recommend removal of all teeth in every patient. This permits the use of larger oral cones or other applicators and results in better treatment.

Radiation Reactions. At the beginning of treatment the patient should be advised what to expect after treatment. He should know that the skin may develop epithelitis, and that mucositis may develop which will make eating and swallowing painful. The effect of radiation on the salivary glands may cause perverted taste and a dry throat for many months, and saliva may become thick and tenacious. The patient should be advised also that a hair-bearing surface may permanently lose its hair. The physician should in all instances refer to these changes as "reactions" and not as "burns."

Antibiotic therapy may lessen the seriousness of mucositis and make eating more tolerable. Severe skin reactions and mucositis usually culminate within 10 days after the total dose of radiation is delivered and then slowly subside. Soaps should not be used, nor should the patient shave over skin showing acute reaction.

Nausea, vomiting and leukopenia are not common following radiation of oral cancers. Radiation ulcers may develop as a delayed reaction and are often mistaken for recurrence of the original tumor. These are usually painful, whereas recurrent tumor is not. Definite diagnosis can be established by biopsy.

Modalities of Radiation Treatment

Many modalities are available and it is important that the proper one be chosen for each individual case of oral cancer. X-ray therapy in high and low voltage, radium, radon, Co^{60}, Ir^{192} and Au^{198} are each used to advantage in the appropriate situation. Low voltage therapy is adequate for surface lesions. High voltages are necessary when deep penetration is required. Radium and the other isotopes can be used in molds for contact therapy. Implants of radon or Au^{198} are frequently used to supplement external high voltage x-ray therapy.

The radioactive isotopes, Co^{60} and Ir^{192}, have recently been used in nylon tubing as interstitial implants. These isotopes and the method of implantation were developed at the Ohio State University Health Center.[4] The radioactive sources are spaced at regular intervals in the tubing. The method of implantation has been described elsewhere.[2] Other advantages offered by radioactive isotopes are their economy and the diminished hazard in their handling. The main use of these radioactive isotopes has been adjunctive to surgery.

SPECIFIC MANAGEMENT

The treatment of oral cancer can be divided into two problems, primary and metastatic involvement.

Primary Lesions

Lips. Controversy remains over the relative merits of surgery versus radiation in the management of carcinoma of the lip. The exponents of radiation therapy state that they can obtain an equal cure rate and that the deformity is less. Since most cancers of the lip are of the squamous cell type, many, especially the smaller ones, can be sterilized by radiation therapy. However, other factors must be considered. Actinic rays from the sun predispose to lip cancer. The lips of a person who has approximately 20 years of active life remaining will continue to receive the same irritation from the actinic rays that they had before the cancer developed, plus the concentrated permanent effects of x-rays.

It is our belief that cancer of the lip is primarily a surgical problem. A good result can be obtained by radiation if the patient is old, but if he is young or if the lesion is large, surgical resection produces better results. The morbidity is less from the cosmetic, functional and curative standpoints. Cancers up to 2 cm. in size can be treated by simple V-excision. Even if the lesion is larger, it lends itself well to wide surgical resection and closure by cheiloplasty. In treating a large

lesion by radiation, the defect must heal by scarring and the cosmetic appearance is not satisfactory.

If a lesion is to be treated by radiation therapy, an adequate total dose should be applied and the teeth and jaws should be properly shielded during this treatment.

Tongue. Cancer of the tongue causes more deaths than any other head and neck cancer. Its treatment depends upon the size, location and type of the primary neoplasm, as well as the extent of metastatic spread. The most common cancer of this organ is squamous carcinoma, a comparatively radiosensitive tumor. Accordingly, the choice of therapy is divided between those who treat by radiation and those who treat by surgery. It is the author's opinion that cancer of the tongue is primarily a surgical problem,[1] but that some form of supplemental radiation therapy is often used to advantage.

Lesions which involve the anterior third of the tongue can be extirpated by obliquely transecting the apex of the tongue. This type of partial glossectomy can be performed easily with only slight impairment of the functions. A rather extensive hemiglossectomy can be performed without altering the patient's speech or ability to swallow to any incapacitating degree.

Malignant tumors involving the dorsum of the tongue are rare, but can be controlled by an elliptical excision of the area. A lesion which involves the posterior third of the tongue and is well lateralized may be approached intraorally. More extensive lesions implicating the base of the tongue are approached laterally because exposure by the intraoral route is not satisfactory. When necessary, the entire base of the tongue can be resected, and the middle third can be recessed and sutured to the base of the epiglottis.

Good results have been reported from treating primary carcinoma of the tongue by radiation. There are disadvantages to this method, however. Many highly differentiated tumors are radioresistant and do not respond well. Patients frequently develop a very sensitive and painful tongue after the maximal dose has been applied and even though the carcinoma is controlled, this creates a lasting problem. Taste is often perverted. Radionecrosis with its attending pain and discomfort occasionally develops.

In our opinion radiation therapy is expedient only when surgical resection is not feasible. For example, to avoid subjecting a patient to total glossectomy, we would implant interstitial radiation in the form of one of the radioactive isotopes in nylon applicators.

Gingiva. Malignant tumors originating in this structure closely overlie bone, and for this reason we prefer to treat them by surgical resection. It is adequate to resect only a small portion of the jaw if the lesion is small, but if it is large and definitely invades the jaw, a

radical resection should be accomplished. As a general rule, radical neck dissection is performed in continuity with resection of the mandible for carcinoma of the gingiva. If the lesion is located in the upper gingiva, a portion of the overlying alveolus is also resected. If much of the maxilla is to be removed, it is better to approach it externally. As soon as healing is sufficient, a soft rubber prosthesis is inserted into the cavity to facilitate talking and eating. Later, a temporary plastic prosthesis is made, and finally an upper denture with a superimposed obturator mound can be constructed.

Palate. Malignant tumors, chiefly squamous cell carcinoma, occur more frequently in the hard than in the soft palate. Less frequently adenocarcinoma originates in the minor salivary glands. The treatment of choice for a lesion of the hard palate is surgical resection, because of the proximity to underlying bone. Squamous cell lesions occurring in the soft palate can be effectively treated by proper radiation therapy. The soft palate lends itself readily to surgical resection, but the functional results are markedly impaired. Surgical treatment is necessary for the notoriously radioresistant adenocarcinomas of the soft palate. Occasionally a very low-grade carcinoma is seen in the mucosa of the hard palate of a patient who wears upper dentures. Clinically, it may resemble marked hypertrophy of the mucosa, but the pathologic sections show carcinoma. Usually this low-grade carcinoma can be controlled by simply removing the mucosa. For other types of cancer of the hard palate it is advisable to remove the bone.

For lesions which involve both the hard and soft palate, interstitial nylon tubing containing radioactive isotopes has been used.

Buccal Mucosa. Tumors of this region are especially important to the dentist because of their relation to dental irritation. Premalignant areas of leukoplakia often develop. If the lesion is small and the diagnosis doubtful, it should be entirely excised to obtain sections for pathologic examination. Most malignant lesions occurring in the buccal mucosa are of the squamous cell type. This area responds well to either radiative or surgical therapy. Good results can be obtained for an older patient by the use of external high voltage therapy, followed by an interstitial implantation. The port should be just large enough to cover the area so that a greater total dosage can be delivered to the tumor. If the patient is young or if the lesion has recurred after previous irradiation, the treatment should be surgical resection. A wide excision can be carried out and the defect closed per primam if only the mucosa is involved. If it invades the musculature, resection must be carried deeply and sometimes it is imperative to remove the cheek. Grafting may be necessary if the defect is too great for primary closure. If a buccal mucosal lesion extends near enough to

the gingiva it will be necessary to resect a portion of the mandible, and this should be dealt with surgically in the same manner as carcinoma of the gingiva. Consultation with a dentist is advised so that any irritative dental surfaces can be corrected.

Jaws. More benign than malignant tumors are found in the jaws. Metastatic lesions from the primary sites anywhere in the body may lodge here. Destruction of the jaws may take place by direct invasion from intraoral cancer or from cervical node metastases. In addition, primary bone tumors may occur in either jaw. The first step in the management of these tumors is to establish proper diagnosis. Often, small cystic lesions which appear grossly benign are curetted without benefit of pathologic study. Fortunately, most of these are simple epithelium-lined cysts which are controlled by curettage. There is always the possibility that it may be an ameloblastoma or, more seriously, cystic degeneration of a malignant tumor. *Regardless of how innocuous it appears, it is important that any tissue removed for the management of a jaw lesion be studied by a pathologist.*

Ewing's sarcoma and reticulum cell sarcoma of the jaw are comparatively radiosensitive and are well treated by radiation. Other sarcomas are competently treated by surgical resection. If the bony involvement represents spread from a primary cancer in the oral cavity, the treatment of course is resection of the primary tumor and a portion of the involved jaw. A segment of the jaw can be resected at the time the radical neck dissection is performed if there is an extension from cervical node metastases. Ameloblastomas usually produce damage by local extension and invasion, and should be treated by wide radical resection. The sarcomas generally spread by a vascular route and very seldom involve lymph nodes. The treatment for sarcomas with encroachment to the mandible is resection of a wide segment of this structure. It is possible to remove a portion of the mandible if the tumor is well localized, and bridge the defect by a bone graft. If the mucosa of the mouth is not entered, which is the exception, it is possible to apply the bone graft at the time of the resection. For malignant tumors, it is often necessary to remove the mucosa immediately covering the mandible as well as the musculature and other tissues in the floor of the mouth. Because of the bacterial contamination that is then present, and the lack of soft tissue necessary to cover such a bone graft, primary grafting is usually impractical. Necessary removal of a portion of the mandibular arch requires some type of fixation to maintain the position between the two ends of the mandible until application of the graft. Vitallium plates may be molded to the contour of the mandibular arch.

It is wise to include at least a submaxillary dissection when resecting

the mandible because of the nearness of the bone to these structures and because the incision often traverses the floor of the mouth and may result in occlusion of the duct to the submaxillary gland. If the submaxillary gland is not removed there may be recurrent swelling later which may be confused with tumor.

Emphasis is placed on adequate surgical resection in tumors involving the maxilla. Exposure is obtained through the Weber-Fergusson approach.[5] The amount of resection depends upon the size of the tumor. If the tumor extends into the infraorbital plate, it may be necessary to exenterate the orbit for adequate resection.

Salivary Glands. Malignant tumors may occur in any of the salivary glands. Patients often associate swelling of the parotid, submaxillary and minor salivary gland areas with dental pathosis and frequently seek the advice of dentists. Early diagnosis is an extremely important phase in the management of these tumors. Because they are usually not "open lesions," the diagnosis should be established by aspiration biopsy. If a formal biopsy is performed, the incidence of spread of the disease is greater and there is less chance for eventual control.

It is ordinarily accepted that the treatment of choice for malignant salivary gland tumors is surgical resection, as most of them are radio-resistant. An adequate excision can be accomplished without much technical difficulty if the tumor originates in the buccal surfaces, the posterior labial surfaces, or in the floor of the mouth. The technique may be somewhat handicapped if the lesion originates in the base of the tongue and it may have to be approached through the neck after resection of a portion of the mandible. A part of the palatal bone must be resected with the tumor if it originates in palatal mucosa.

The feel and fixation of a lesion should aid the surgeon in his critical evaluation of whether a lesion is benign or malignant. A tumor with indiscrete margins, as often occurs in the palate, requires wider resection even though histologically the lesion is benign. Benign salivary gland tumors are usually discrete and movable.

Dissection of the submaxillary triangle with removal of the entire submaxillary gland and tumor should always be performed for management of malignant tumors of this gland. With care, the lingual and hypoglossal nerves may be preserved unless they are jeopardized by the proximity of malignant tissue. After the submaxillary procedure has been completed an entire radical neck dissection is performed if there are no medical contraindications.

The treatment of choice for malignant tumors of the parotid gland is total parotidectomy. The facial nerve warrants special attention in the execution of this procedure. If the tumor is located so that it does not compromise the common facial nerve or its branches, the parotid gland may be removed without producing any paresis. On the other

hand, if there is obvious malignant tissue proximate to any part of the nerve, it must be sacrificed to control the tumor.

Mucoepidermoid carcinomas and malignant mixed tumors of the parotid gland grow locally and seldom metastasize. They can be well controlled by parotidectomy. On the other hand, adenocarcinomas metastasize more frequently and are treated by parotidectomy with neck dissection.

Irradiation may be used for malignant salivary gland tumors if surgery is medically contraindicated. Some form of radiation therapy may also be expedient if during surgery it is found that complete removal of the tumor is impossible. In this instance, nylon applicators containing radioactive material have been extremely practical.

Metastatic Cancer of the Neck

All malignant tumors of the oral cavity have the potential to metastasize. Tumors of various organs have certain predilection for metastasis to the neck but, in general, any oral cancer can metastasize to any lymph nodal area in the neck. The degree with which the various cancers metastasize is not constant. Approximately 10 per cent of all cancers of the lip metastasize during their course, but about 60 per cent of all cancers of the tongue will involve cervical nodes.

Surgery and radiation are available to treat these metastases. For the uncomplicated metastatic lesion of the neck with a primary lesion controlled, the ideal treatment is radical neck dissection. If the primary lesion has not been treated, then it is advisable that surgical removal of the primary tumor be accompanied by neck dissection. Some form of radiation therapy should be used for non-resectable tumor masses.

Surgery. Metastases to nodes in the neck require radical neck dissection, regardless of the level of involvement. This is a surgical procedure designed to remove the lymphatic tissue between the clavicle and the mandible en bloc.[3] If the primary oral cancer has not been controlled by previous therapy, the radical neck dissection is performed in continuity with resection of the primary tumor. It may not be necessary to resect mandible unless the tumor is proximal to bone. With the cheek flap properly retracted and the mandible transected, adequate exposure of any part of the oral or pharyngeal cavity is obtained and the resection can be carried out under direct vision. The resulting deformity depends upon the amount of bone removed. Prior to such surgical procedures it is advisable to have dental consultation with reference to the extraction of teeth and periodontal disease and dental hygiene.

Radiation. Occasionally radiation therapy is advocated as the principal treatment for involved nodes when they can be treated

simultaneously with the primary lesion. For example, if a carcinoma of the tonsil is irradiated, a high jugular node on that side may be treated at the same time. To insure optimal control, this treatment is supplemented with interstitial implantation. If this program fails to control the metastatic process, the patient is then subjected to radical neck dissection. Other than this, radiation therapy for metastatic cancer of the neck is used primarily as an adjunct to surgery. This may be either high voltage external therapy or implantation of interstitial radioactive isotopes.

Biopsy Technique. It is very important that the exact diagnosis be established before treating any node in the neck. Biopsy procedures of the neck deserve special note. Local spread of disease or production of scar tissue may result from the execution of an incisional biopsy. Further surgical resection is limited if the scar tissue is in the area of a vital structure, such as the carotid artery. Aspiration biopsy is the procedure of choice in establishing diagnoses of masses in the neck. It is a short, simple office procedure performed under local anesthesia without morbidity, and the accuracy of diagnoses is very high. The area to be aspirated is first sterilized and then locally anesthetized. A small incision is made in the skin with a pointed blade to prevent the inclusion of surface epithelium. A 16 or 18 gauge needle is introduced; a stylet precludes the inclusion of extraneous tissue. After the center of the mass is reached, the stylet is removed and a syringe is applied for suction. By moving the needle to and fro an attempt is made to core out a small portion of the tumor. A formal incising biopsy is employed only if the diagnosis cannot be established by needle aspiration. In a recent report by the author on the management of metastatic neck cancer, aspiration biopsy established a diagnosis of malignant tumor in 96 of 101 attempts.

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Postsurgical Prosthesis

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Increasingly effective therapy for oral and facial cancer is presenting the dental profession with a correspondingly increased opportunity and responsibility for rehabilitating these patients.

The passing of time and additional experience reveal a recurrence of relatively typical problems that are not too difficult for treatment by any good dentist. These cases we believe to be a responsibility and field of real service for the man in general practice, the family dentist. Basic problems of this type will be discussed here, plus suggested techniques for prosthetic treatment.

THE EDENTULOUS MAXILLARY ARCH

Following cancer therapy, openings of various sizes are not uncommon in the edentulous upper arch. Regardless of the size of the aperture, the problem is to close it in order (1) to improve speech or, in some instances, to make speech possible; (2) to prevent food from reaching the maxillary sinuses and nasal cavities; (3) to reduce the flow of exudates into the mouth; and (4) if possible, to restore at least some masticatory function. The object of the impression making should be to include all of the area that can be covered with a complete denture, and to provide for the positioning of an obturator in the opening. Within the limits of tissue tolerance and elasticity, the obturator should, if possible, extend far enough into the opening to afford a "buttoning in" effect. This would accomplish some additional retention for the prosthesis. Frequently, surgical treatment of oral cancer involves the removal of one maxilla, and with it the floor of the orbit and the eye.

After healing, it is recommended that, initially, the oral prosthesis be made only for the purpose of closing the opening and restoring the form of the edentulous maxillary arch.

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Make a preliminary impression with either modeling compound or alginate.

With compound, care must be exercised in contacting areas sensitive to pressure or temperature. Be careful also not to use it in such excess that its removal after hardening would be very painful, or even impossible, as a result of extension too far into undercut or displaceable areas.

If modeling compound has also been selected for the final impression, the preliminary one may be modified and perfected by flaming and/or adding compound until a satisfactory result is obtained. Additional refining may often be accomplished by the addition of impression wax in critical areas.

With alginate, if the opening is large, the stock tray should be blocked in well enough to carry the alginate to the area desired. The cavity should be filled with gauze to the approximate depth to which you feel the tissue displacement will finally allow the prosthesis to be inserted. Coat the gauze well with petroleum jelly. Unless there is ample access for removal of the gauze, be sure to tie a string to it, with a long enough end available, to facilitate removal.

This primary impression is then used to form a cast upon which a more accurate tray may be fashioned for making the final impression. If the preliminary cast is thought too shallow in the opening it can be scraped to a greater depth. If it is too deep, it can be blocked in.

If alginate is chosen for the final impression a suitable tray may be prepared from base plate material or self-curing acrylic resin with enough holes drilled into it to provide retention for the alginate.

The resulting cast (Fig. 1A) is waxed over with a thickness of approximately 3 mm of base plate wax, including the portion against the inside of the cheek on the side with the opening. It is waxed into the buccal and labial vestibules on the side of the remaining ridge to the maximum possible, as for a complete denture (Fig. 1B).

In the region of the opening, build up on top of the wax, with any satisfactory spacing material, the approximate form of the lost portion of the ridge (Fig. 1A). Ordinary window caulking compound is practical for this step. Build it up to within about 3 mm of the contour of the natural ridge. Now wax a separate lid of base plate wax to complete the form of the ridge and to fit accurately to the original layer of wax (Fig. 1C). Remove this lid and the spacing material. Flask the cast with the first layer of wax, and process in clear acrylic resin. Also flask and process the lid in the same material.

Deflask and polish both acrylic parts well. Try the base in the mouth, and check for any undue pressure areas, as with any template. When satisfied with the fit, attach the lid to the base with self-curing
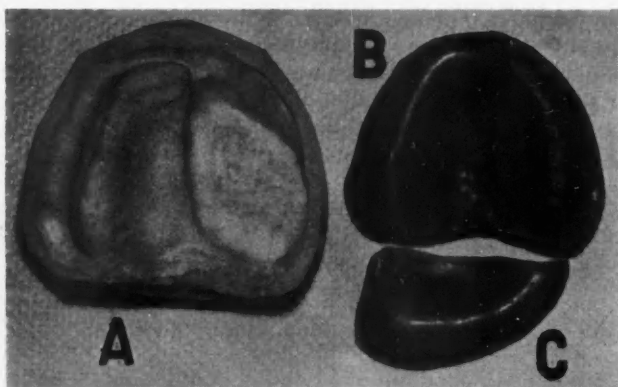
Fig. 1. Edentulous arch. *A*, The final cast. *B*, Base wax-up with spacer. *C*, Wax-up of lid.

clear acrylic and polish well. The resulting appliance should now restore the edentulous ridge form. It is as light in weight as possible since the portion which fills the opening is hollow. The restoration of the ridge will permit adequate speech, aid in eating (with the help of the tongue), prevent food from entering the cavity, and restrain exudates. The clear acrylic permits subsequent additional checking for undue pressure.

Such an appliance should be worn for at least a year, pending possible recurrence of the malignancy. Only then, if at all, may a denture be constructed or restoration of the occlusion attempted. If this is decided upon, the same space as was in the obturator may be built into the denture. If the aperture is due to a cleft palate or other malformation, a denture may be attempted as the original prosthesis.

The operator should be aware of the great service rendered the patient by the obturator alone, even if a denture is not attempted later. It should also be obvious that this is not a difficult procedure.

THE PARTIALLY EDENTULOUS ARCH

In the partially edentulous case the objective is to close the aperture, using the teeth for support and retention by employing enough of them to distribute the load as widely as necessary, and thereby preserve those abutments as long as possible (Fig. 2A).

The preservation of the remaining teeth demands serious consideration of the advantages of full coverage for them. This procedure should minimize the possibility of caries and abrasion under clasps, and it usually permits better clasping contours to be developed.

Finally, multiple clasping of teeth (in excess of the requirements of the usual partial denture) is a safeguard against the future loss of one or more abutments, as well as a means of distributing the load over more abutments (Fig. 2B). If any loss of abutments should occur, sufficient clasping remains to make unnecessary the reconstruction of the denture. The space left by a lost tooth can readily be filled with
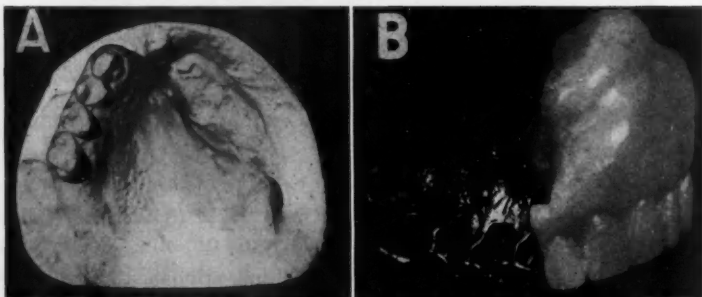
Fig. 2. Partially edentulous arch. A, The cast. B, Appliance with multiple clasping.
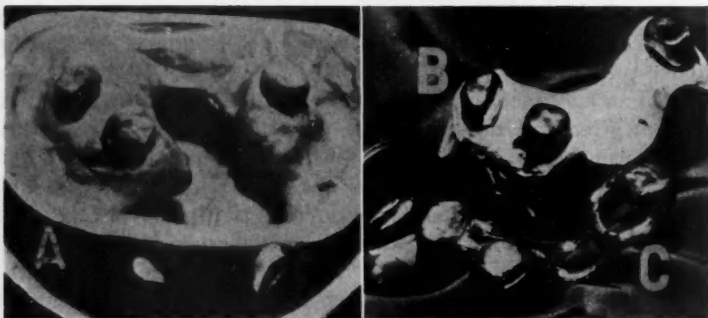
Fig. 3. Partially edentulous arch, malpositioned teeth. A, The cast. B, Inner shells of telescope crowns. C, Outer shells with cervical retentive arms.

a tooth and base material and the remaining clasping can be relied upon to give adequate support.

With only six teeth or fewer remaining, the author would generally advise full coverage and clasping for all, in the presence of an aperture of any appreciable size (Fig. 2B).

Sometimes, after surgery, quite stable teeth are present with a portion of root exposed (Fig. 3A). They should be retained as long as possible as abutments by including coverage of the exposed root through extension of the crown (Fig. 3B). The impression for the

crown can be accomplished by the material of your choice. However, the tray or tube for the impression must be prepared in such a manner as to support and confine with some pressure the material used, in order to assure good adaptation at the periphery.

For elastic impression materials a dammed and blocked out bridge tray will usually fill these requirements. For a rubber impression material it is important that reliable rubber adhesive first be painted inside the tray. Copper band impressions are not recommended for elastic materials because of lack of bulk, difficulty in getting the band long enough to cover the exposed root, and distortion of the band under pressure followed by its springing back when removed.

Any comparatively complete clasping is acceptable with the thought in mind that should a tooth be lost the remaining clasps and rests would still function to maximal effectiveness. Since maximal assistance to ridge and soft tissue is desired, relatively rigid clasping is indicated and bar type clasps would normally only be resorted to through necessity (Fig. 2B).

MALPOSITIONED ABUTMENTS

The third basic problem, that of malpositioned teeth, is present in cancer cases where the number of remaining teeth or their position contraindicates clasping and in many cleft palate or malformation cases (Fig. 3A). The treatment usually involves full coverage with some type of telescoping crown. The resulting prosthesis has the appearance of a full denture, with the functioning teeth in proper relation to the ridge and the opposing arch.

Three basic treatments are possible. One of these is the use of simple telescopic crowns for the abutments. The abutments are prepared for full coverage, the inner shells are tapered only slightly in conformity with each other (and with any tissue undercuts) to permit a common line of insertion of the telescoping outer shells, and the inner shells are cast and polished.

If the outer shells are to be used only as support, buried inside the denture base, they are cast as simple shells to fit over the inner ones, providing frictional retention. If the abutment is positioned so that it may be part of the functional occlusion, then the outer shell would be carved like any full crown and take its place in the set up of the denture teeth. The outer shells may be cast in one piece with the framework (Fig. 3C), or cast separately and soldered to it.

Usually there are only scattered teeth available and the object of the full coverage again is to preserve them as long as possible and to utilize all whose stability warrants.

A second solution, if frictional retention alone is considered insufficient, is to incorporate a cervical retentive arm on one side of each crown (Fig. 3C). The inner shell is prepared with a very slight groove or an undercut at the cervical area (Fig. 3B). The principle of opposition of retentive arms should not be overlooked, i.e., all retentive arms may not be on the same side of the abutment teeth or retention will not be accomplished.

Since the outer shell will be buried in acrylic, provision must be made for the retentive arm to flex. This can be provided for by a shim of stainless steel band material outside the arm, or by painting on a layer of oxyphosphate of zinc cement after the flask is open. With the shim method a portion of the shim is left extended into the investment when flasking and is then pulled out with pliers after processing. When cement is used it can be easily picked out while flexing the clasp inward, or by soaking in hydrochloric acid.

The third possibility for abutments is one favored by some operators when the remaining teeth are considered usable as abutments, but are either too few in number or not stable enough to permit rigid clasping. The method is that of eliminating the frictional retention of a true telescope crown. The tooth stump and inner shell are prepared with a rounded occlusal surface and the outer shell is etched inside with aqua regia to eliminate frictional retention. The rounded abutment effects transmission of occlusal and masticating stresses to the long axis of the tooth. This type of appliance admittedly requires greater use of the tongue and manipulation by the patient to retain it. Its advocates feel that this is justified by the reduction of strain and torque on the abutment teeth and a resulting longer life for them.

SUMMARY

The three basic problems discussed—the edentulous arch, the partially edentulous arch with abutments in normal position, and the partially edentulous arch with the abutments in malposition—comprise the bulk of the cancer patients the general practitioner will see. The techniques described, plus possible modifications by the operator, are well within his capabilities. It is hoped that he will provide this much-needed service to the increasing number of patients seeking it.

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Prosthetic Restoration of Facial Defects

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In recent years there has been an increase in radical surgery on the head and neck, resulting from more effective treatment and earlier diagnosis of cancer. Consequently there are many people alive today who need facial prostheses so that they may be able to take their places in society again, be accepted by others, and possibly become self-sufficient.

Patients have presented the following conditions requiring facial prosthetic restorations: (1) the entire nose or part of the nose missing; (2) the nose missing with facial extensions involving the cheek, or with sinus or intraoral involvement; (3) orbital exenteration with or without nose, cheek, sinus, or intraoral involvement (Figs. 1 and 3); (4) those in groups (2) and (3) who have also undergone excision or partial excision of the mandible, maxilla or condyle (Fig. 7).

With such a wide variety of conditions, it is necessary to have a cooperating team consisting of a reconstructive and plastic surgeon, a head and neck surgeon, a prosthodontist and a prosthetic technician in order to offer a successful program to rehabilitate these people. The material for prosthetic rehabilitation must be economical in cost, in time required to complete a prosthesis, and in the type of laboratory equipment required to process it, and it must be well tolerated by the body's tissues and esthetically satisfactory.

MATERIALS

Polyvinyl chloride, a soft rubbery, plastic material, is quite satisfactory for facial prostheses. It can be obtained as a powder and liquid similar to methylmethacrylate. The powder consists of polyvinyl chloride, 6.5 parts by volume; calcium stearate, 1 part by volume (this acts

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as a heat stabilizer); and a small amount of zinc oxide which acts as an opacifier. The liquid, an oily organic fluid, is dibutyl phthalate and it is usually mixed with the powder in a 1:1 ratio. Adding more liquid will result in a softer, more elastic prosthesis. The polyvinyl chloride mix can be colored to simulate the skin color of the patient by adding oil-soluble dyes which have been dissolved in the dibutyl phthalate.

The material can be processed in inexpensive molds at temperatures readily available in laboratories. Highlights and shadows can be added to the finished prosthesis.

A commercial material known as Flexi-Derm* is available and facilitates the work of the prosthetic technician. This material is probably a mixture of polyvinyl chloride and polyvinyl acetate in solution with dibutyl phthalate.

The Flexi-Derm kit consists of about six basic flesh colors (1 lb. jars containing powder, liquid, and pigment well dispersed); a jar of mixed opaque white; and a jar of mixed "clear." There are about fourteen 1 oz. jars of Flexi-Derm which contain pre-mixed powder and liquid with a concentrated amount of oil-soluble dyes which can be added to the basic flesh colors to further modify the basic shades as required. The kit also contains six ½ oz. jars of dry, oil-soluble dyes which can be mixed with the liquid and used to add any delicate shading and highlights desired.

TECHNIQUE

Preparing the Patient. When the patient is first seen, examine the facial area in need of a prosthesis to determine what can be done to improve his appearance. Some of the questions that should be answered are: (1) How long has it been since the last surgical procedure? (2) Has the area healed sufficiently to withstand some rubbing action? (3) What are the possibilities of recurrence of the tumor or sloughing of a skin graft? There have been cases of recurrence and sloughing even before the prosthetic restorations could be completed.

If the results of this study are favorable, determine the facial area to be restored and outline it on the face with an indelible pencil. Fill severely undercut areas, such as an opening into the sinus or into the orbital cavity, with petrolatum-soaked gauze (Fig. 1). Retain some slight undercuts to help in the retention of the finished prosthesis.

A full face impression and positive cast may be desired by the sculptor, in which case provision must be made so that the patient

* Nelson Kramer Corp., Royal Oak, Michigan.

can breathe through a small flexible tube which can be placed either in the mouth or in the nostril. However, satisfactory results can be obtained with less than a full face impression and positive cast. In either instance, lubricate the facial area (including the eyelashes and eyebrows) with a thin layer of mineral oil.

Taking the Impression. Any acceptable impression material and impression technique can be used as long as the end result is satisfactory. The writer has used impression plaster, "soluble" impression plaster,* alginate,† and hydrocolloid impression materials. Best results have been obtained with soluble impression plaster and, secondly, with alginate.

Fig. 1.   Fig. 2.

Fig. 1. Patient with ocular exenteration which has involved sinus, maxilla, and condyle. The cavity has been filled with gauze prior to taking impression.
Fig. 2. Same patient with orbital-facial prosthesis.

Soluble impression plaster and water sets up in approximately 3 minutes after the mixing begins. If additional setting time is required, a small amount (10 to 25 per cent) of normal setting plaster can be added to the soluble plaster before the water is added.

Apply the soluble plaster-water mixture to the facial area in two layers. First, apply a thin or watery mix as a homogeneous layer having a thickness of about ⅛ inch. If a thicker layer is applied, the weight of the mixture will displace, distort, and compress the facial surfaces. Apply the second layer of the soluble plaster-water mixture, about ½ inch thick, immediately after the initial set of the first layer.

When alginate impression material is used, prepare a tray to carry

* Ransom and Randolph Co., Toledo, Ohio.
† Jeltrate elastic impression material, L. D. Caulk Co., Milford, Delaware.

the impression material to the facial area. A suitable tray can be made with an aluminum sheet (preferably perforated), which can be obtained in most hobby stores. A more accurate fitting tray can be made by using a quick-setting, self-curing acrylic tray material. Mix the tray material according to specifications, roll it to a thickness of ⅛ to 3⁄16 inches and place it over the facial area which has been covered with a ⅛ inch thickness of moist paper. This will allow a uniform thickness of impression material when the final impression is made.

Mix the alginate material with water, following the recommendations of the manufacturer. Pick up some of the mixed alginate with the fingers and place it in the undercut areas of the face to displace air pockets, then place the remainder of the alginate into the tray and place in contact with the facial area.

Pouring the Cast. The soluble plaster impression must first be coated with a separating medium such as liquid soap or any commercial separating medium. Carefully vibrate a thick mix of hydrocal (dental stone) and water into the impression, pouring sufficient hydrocal into the impression to obtain a rugged cast.

After the hydrocal has hardened, place the soluble impression and the hydrocal cast in water, bring to boiling temperature, and leave in the boiling water until the impression disintegrates.

It is necessary to pour up any alginate impression with hydrocal-water mixture immediately after the alginate hardens, owing to the characteristic dimensional instability of the alginate. Separating the elastic alginate impression from the hardened cast does not present any problem.

Making the Wax Prosthesis. Trim the hydrocal cast to make it somewhat symmetrical and rub its surface with talcum powder so that the wax will not stick to its surface.

Adapt a sheet of hard dental base plate wax on the surface of the cast which is to receive the prosthesis to insure a smooth surface on the tissue side of the restoration. The prosthesis can be developed on this with base plate wax.

Designing a satisfactory prosthesis in wax calls for artistic ability, some experience in sculpturing, and knowledge of facial anatomy. If the facial defect is not extensive, it will be a fairly simple matter to make an esthetic restoration. If the defect is extensive, photographs and suggestions from the patient and relatives may be helpful. The technician's artistic ability in large part will determine the cosmetic result.

In waxing up a nose, it is advisable to make the nostrils solid, since a simple two-piece mold can be used. Nostril openings can be made later in the finished plastic nose.

Fig. 3.   Fig. 4.

Fig. 5.   Fig. 6.

Fig. 3. Patient with part of nose missing. Also involved are the eye and sinus.

Fig. 4. Prosthesis made up in wax. The skin texture is quite evident.

Fig. 5. Prosthesis completed in Flexi-Derm material. Eyelashes and eyebrow have been restored.

Fig. 6. Patient with the completed prosthesis. Photo-flood lamps which were not color corrected were used to take this picture, thus prosthesis looks dark.

If the prosthesis is to contain an artificial eye, the facial prosthesis should be made in two pieces, a front portion and a back portion which will support the artificial eye, because processing the facial prosthesis would damage the artificial eye whether it is made of plastic or glass. The two sections can be fused together when completed in plastic.

Adapting the Wax Prosthesis to the Patient. The wax prosthesis must be adapted to the patient, since invariably the impression will have some minute discrepancy. Submerge the entire wax prosthesis in warm water (95° to 105° F.) until it becomes slightly plastic, then place it on the patient's face and carefully adapt it to the tissue. The

wax restoration can now be modified so that a more esthetic restoration can be made.

The final step in completing the wax prosthesis is to provide a means for its retention. If there is nasal involvement, make a bed on the wax nose to accommodate the spectacle bridge and nose pads. This type of retention has proven to be especially satisfactory if a sturdy, all-plastic spectacle frame is used.

Preparing Basic Skin Shades. Before the patient is dismissed, prepare the Flexi-Derm skin shade required for the patient. Small samples of mixed shades can be processed quickly by picking up a small quantity of material with an applicator stick and immersing it into a beaker of hot glycerine (300° F.) for 3 minutes.

Select a pre-mixed basic skin shade that is slightly lighter in shade than that which is required, and add small quantities of the concentrated mixed Flexi-Derm pigments. Each time a change is made, cure a sample of plastic and check for color match. Repeat this until a satisfactory shade is obtained. The basic shade can be made more opaque by adding the "opaque white" material, or more translucent by adding "clear" material. Check the colors in daylight, for artificial light is misleading.

Making the Mold. Dental refractory materials such as those used to cast gold inlays or partial dentures will make a satisfactory mold material for Flexi-Derm. The following mold materials have been successfully used:

> 20% Hydrocal,* 80% Kerr model cristobalite†
> 20% Hydrocal, 80% Kerr inlay cristobalite investment†
> 20% Hydrocal, 80% Ticonium investment‡
> 20% Hydrocal, 80% R & R gray investment‡

The hydrocal gives the mold material strength, while the refractory material conducts the heat to the plastic and prevents the mold from disintegrating at the high curing temperatures.

Select a dental flask large enough to contain the wax prosthesis (Figs. 9 and 10). Make a thick mix of one of the above combinations of hydrocal, investment and water, place the mix in the lower half of the flask and settle the wax prosthesis (the tissue side against the investment) on the investment. Do not submerge any of the margins of the wax prosthesis under the investment.

Applying Skin Texture to the Wax and Completing the Mold. After the investment material in the lower half of the flask hardens, clean

* Coecal, Coe Laboratories, Inc., Chicago, Illinois.
† Kerr Manufacturing Co., Detroit, Michigan.
‡ Ransom and Randolph Co., Toledo, Ohio.

Fig. 7. Fig. 8.
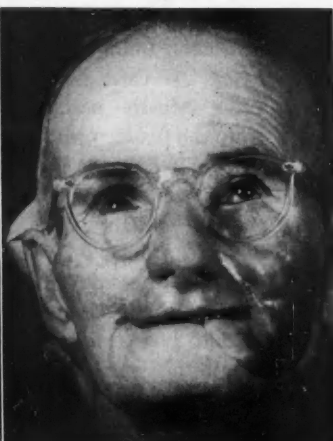
Fig. 9. Fig. 10.

Fig. 7. Patient with part of nose and lips missing. Also involved are the sinus, part of mandible, and part of the maxilla.

Fig. 8. Same patient with facial and dental restoration.

Fig. 9. The refractory investment mold. Note the metal dowels and sleeves used to maintain the proper relationship between the two halves of the mold.

Fig. 10. An improvised tin flask used to contain the refractory mold shown in Figure 9.

the exposed wax surfaces and develop the skin texture (Fig. 4). This may be done by stippling the wax surface with a stiff bristle brush and then flaming the wax gently, or by making pits in the wax with a sharp instrument, then flaming the wax gently. It should be remembered that the pores are deeper and closer together in some places than in others.

After simulating the skin texture on the wax, apply a separating medium on the investment and very carefully pour up the upper half of the mold. Air bubbles in the investment adjacent to the wax will ruin the appearance of the prosthesis.

Preparing the Mold for Packing. The mold may be opened when the flask becomes warm owing to the heat of reaction of the setting of the investment. Remove the softened wax and rinse the mold with boiling water. Use care in wax removal to prevent the wax pigment from entering into the pores of the mold, as this pigment could be transferred to the plastic prosthesis during processing.

Apply one or two coats of Kerr's Glossy-Glaz* to the surfaces of the investment and allow it to dry. This will act as a separating medium and prevent the plastic from sticking or fusing to the investment during processing.

Packing. Warm the empty mold in a dry heat oven at 190° to 200° F. until the mold comes up to temperature, then place the mixed Flexi-Derm into both sides of the mold and place both halves of the mold back into the oven. The mold heat plus the oven heat will cause the plastic to thicken in 5 to 10 minutes.

After the plastic has thickened, place wet cellophane between the two halves of the mold and make a trial closure. If sufficient material is in the mold, final closure is indicated. The pressure required for trial closure and final closure is very slight, usually that which can be obtained by tightening the flask with a C-clamp by hand.

Processing. Submerge the mold, held closed with the C-clamp, into a hot glycerine bath (260° to 280° F.) for approximately 25 to 35 minutes. Higher temperatures which would require less processing time could be used, but the life of the mold will be reduced.

Overheating or heating the mold too long will cause the plastic to become darker, scorched, and harder than required. However, processing a few minutes more or less on subsequent curing cycles will produce shades that are slightly darker or slightly lighter, respectively. Thus slight variations can be obtained in the basic skin color with the same batch of material.

After processing, cool the mold at room temperature for about 10 minutes. The cooling rate can then be increased by using a fan or dipping the flask in cool water. Rapid thermal change will damage the mold.

Finishing the Prosthesis. Remove the prosthesis from the mold carefully so as not to damage the mold or stretch or distort the plastic restoration. Remove the flash with a sharp surgical scissors, surgical scalpel, or mounted rotating stone burs. Use care so that the skin texture is not destroyed in the finishing process.

Eyelashes can be sewn onto the lids very easily by using an ordinary needle and human hair which has the proper texture and the proper

* Kerr Manufacturing Co., Detroit, Michigan.

color, noting the quantity of lashes needed (Fig. 5). The eyebrow can be simulated by pushing hair into the Flexi-Derm at the proper angle with a very small inverted U-shaped instrument (a needle with half of the "eye" ground away is quite satisfactory). Small quantities of human hair having various characteristics can be obtained from wig makers or from donors.

If the prosthesis is made in two sections (in order to incorporate an artificial eye), the two sections may be fused together by placing them together in their correct relationship, applying a very small quantity of uncured Flexi-Derm at the junction where the two layers make contact, covering that area with a piece of dry cellophane and applying a hot wax spatula to the area. The heat will cause the two pieces to fuse together and the cellophane will prevent scorching of the prosthesis. This operation is repeated until the entire periphery has been fused.

Highlights and shading can be adroitly added to the prosthesis superficially by painting very dilute solutions of oil-soluble dyes and dibutyl phthalate. The dye solutions can be applied with a brush or with cotton swabs. Several applications may be made if necessary, but it is most difficult to remove any of the pigment if the concentration is too great.

Retention of the Prosthesis. Most patients dislike permanent attachment of spectacle frames to a facial prosthesis for retentive purposes. Whenever possible, provide frictional retention between spectacles and prosthesis so that the patient may remove either the spectacles or the prosthesis independently.

Use undercuts and surgical adhesives° to assist in the retention of the prosthesis. Many patients prefer to use narrow strips of the tape, adhesive on both surfaces, which is used for retaining wigs. This is more desirable than using paste-type adhesives. Place the adhesives on the portion of the prosthesis that contacts the healthy facial tissues.

DISCUSSION

The technique presented in this article is quite similar to that employed by others, the main differences lying in (1) employing refractory investment molds for processing, and (2) the processing technique itself.

The refractory molds have been used as many as six times before

° Surgical adhesives: Adhesive No. 209, Vernon Benshoff Co., Pittsburgh, Pennsylvania; Duo Liquid Adhesive, Johnson & Johnson; United Skin-Hesive, United Surgical Supplies, New York, New York; Flexi-Derm Adhesive, Nelson Kramer Corp., Royal Oak, Michigan.

damage became apparent. One of the molded pieces can be used to make another mold if necessary. After several years, if a patient requires a new prosthesis, either the old prosthesis or an unused prosthesis can be re-adapted by adding soft wax to the tissue surface to correct any discrepancies due to changes on the face, or the old prosthesis can be modified to bring about any desired improvement. Then a new refractory mold can be made.

This technique eliminates the need for making and storing expensive permanent metal molds which may make an ill fitting prosthesis several years later.

Polyvinyl chloride materials (of which Flexi-Derm is one) are satisfactory in many respects, especially in simulating the skin colors and in the relative ease with which they can be used to make prostheses. Flexi-Derm saves considerable time in the formulation of the proper skin shades by providing pre-mixed and pre-pigmented materials. Unfortunately, these materials do not have as ideal physical properties, and some of the prostheses, of necessity, have been made a little thicker and heavier than desirable in order to have sufficient strength and tear resistance.

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Foreword

Endodontics is a most satisfying branch of dental practice. It is gratifying to save a useful organ for years of service to an appreciative patient. The prognosis of a properly treated and filled root canal is more favorable than a coronal restoration or a tooth replacement because it is affected only by the internal environment. In restorative dentistry the variable influences of the external environment, such as saliva, diet and trauma, are unavoidable hazards.

The carefully treated root-filled tooth is no longer regarded as a menace to health. Unquestionably, the inadequate, often careless techniques that were widely employed to treat pulp-involved teeth prior to the development of the concept of focal infection contributed to infection in the jaws. While focal infection was greatly overstressed immediately after its conception and many pulpless teeth were needlessly extracted, it stimulated a vast improvement in the techniques of treatment. The consensus today is that demonstrable infection should not be allowed to remain in the body, but that the root-filled tooth that has been in the mouth for some time with no evidence of disturbance in the surrounding tissues does not jeopardize the health of its host.

Techniques may vary considerably in detail and yet accomplish the same successful result. However, there are certain fundamental principles that must underlie endodontic treatment if the best results are to be obtained. Success or failure depends on the tissue reactions that occur in the apical region of the tooth. If the resistance and reparative powers of these tissues are high, favorable results usually are obtained. We all have seen cases that have succeeded despite the treatment, thanks to nature's generosity. On the other hand, when the natural resistance of the tissues is low, failure is likely to occur.

We have no means of measuring the tolerance and the reparative potentialities of the apical tissues at the time of treatment. The apical tissues must of necessity be traumatized at the time of vital pulp amputation and they usually are disturbed in those teeth selected for

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treatment that have necrotic pulps; our technique should not add unnecessarily to the existing injury.

There are five cardinal procedures essential to satisfactory endodontic treatment. The first procedure is diagnosis and treatment planning. After it is determined that the condition of any tooth indicates that the preferred form of treatment is root canal therapy, only those patients for whom a favorable prognosis can be made should be selected to receive such treatment. The second procedure is thorough mechanical cleansing and enlarging of the root canals; the third, complete and permanent filling of the root canal as far as the operation extends, preferably to the dentino-cemental junction. The fourth procedure is the establishment of surgical cleanliness, and the fifth, the administration of proper medication.

The contributors to this symposium on endodontics have discussed and illustrated the fundamental principles and procedures. We appreciate their cooperation and the effort that they have made to give the practitioner a useful description of modern endodontic practice and to help him solve the problems related to it.

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Selection of Teeth for Root Canal Treatment

CHARLES G. MAURICE, D.D.S., M.S.[*]

Correct diagnosis is a primary requisite in selecting cases for root canal therapy. The treatment of choice for pulpally involved teeth which have functional and esthetic value is root canal therapy rather than extraction. Nevertheless, it must be realized that limitations to this form of treatment exist and that root canal therapy is neither always possible nor always indicated.

The claim that root canal therapy is too difficult for the general practitioner or that the possibility of failure is too great is contrary to fact and can no longer be accepted. Fortunately, dentistry as a whole in recent years has adopted a rational and realistic attitude in regard to root canal therapy. This in large measure has been the result of significant progress in the biologic phase of dentistry.

The decision to treat or extract the tooth is usually reached after clinical signs, roentgenograms, and the patient have been studied. The general practitioner who wishes to avoid complications may find it judicious to limit root canal therapy to teeth which lend themselves favorably to treatment whereas the endodontist can exercise greater latitude.

It is difficult to formulate hard and fast rules which always apply in reference to teeth which can or cannot be treated. Each case must be considered individually and many factors must be appraised. Variables introduced by both patient and operator can markedly influence prognosis. For example, the skilled dentist might be able to cope satisfactorily with certain mechanical complications which the less proficient or less experienced operator might find insurmountable; a tense and uncooperative patient can sometimes complicate treatment to the extent that competent treatment cannot be carried out.

From the standpoint of ease of treatment, teeth ordinarily fall into two broad groups. The first group includes teeth which as a rule are

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easy to treat, and the second group includes teeth where some difficulty in treatment may be expected.

Teeth relatively simple to treat include (1) teeth with one root; (2) teeth readily accessible; (3) teeth with fully formed roots and straight, open canals.

Teeth generally more difficult to treat include (1) teeth with more than one root or more than one canal in each root; (2) teeth not easily accessible; (3) teeth with narrow, partially obstructed, curved, or bifurcated canals; (4) teeth with unusually wide canals which are open at the apex; (5) teeth with fractured or badly destroyed crowns.

The degree of difficulty encountered in treating teeth belonging to the second group will depend to a large extent upon the manual skill, clinical experience, and scientific knowledge of the operator. It should be evident therefore that advisability of root canal treatment varies with the dentist. Nevertheless, some conditions nearly always contraindicate root canal treatment. Knowledge of these will help the dentist to recognize cases in which little or no likelihood for success exists. From the standpoint of eliminating failure in root canal treatment, it is extremely important to know when not to treat. Furthermore, since contraindications for root canal treatment apply to all, they can be readily listed. A major portion of this article will be devoted to a discussion of valid contraindications to root canal treatment and to some conditions in which advisability of treatment is controversial.

CONTRAINDICATIONS TO ROOT CANAL TREATMENT

The defense mechanism and healing potential of the tissues of the patient play an important role in the success or failure of root canal therapy. Conditions local or systemic, which seriously interfere with the capacity of periapical tissues to defend themselves from injuring agents or to heal, contraindicate root canal therapy. Conditions in this category are less frequently encountered than many dentists imagine.

Routine root canal treatment is contraindicated in teeth which present the following local conditions:

1. Teeth with radicular (periapical) cysts.

2. Teeth whose root canals contain necrotic material which cannot be removed because of calcifications, packed debris, or broken instruments; because the canal is unusually curved or tortuous; or because the root is fractured.

3. Teeth with a fistulous tract connecting the periapical region with the gingival crevice.

4. Teeth whose roots have been mechanically perforated at a level

below the epithelial attachment (an exception to this rule will be considered later).

5. Teeth in which the side of the root has been perforated by pathologic internal or external resorption.

6. Teeth with gross incomplete development of the roots in which the pulp has died.

Radicular Cysts

Radicular cysts contraindicate root canal treatment because they do not resolve after root canal therapy. Since they tend to grow continuously at the expense of bone they should be enucleated by surgery at an early stage. Radicular cysts when sufficiently large can deform the face, cause pathologic fracture of the jaws, and other complications.

Irritants inside the root canal undoubtedly play a major role in the initiation of radicular cysts. The reader is referred to any good textbook on oral pathology for a description of the manner in which radicular cysts develop and the classification into which they fall.

Clinical experience has shown that radicular cysts cannot be positively differentiated from other periapical conditions associated with pulpless teeth by means of roentgenographic or clinical examination alone.

Pulpless teeth may give rise to a number of conditions other than radicular cysts which cause periapical loss of bone. Root canal therapy can effect healing in all such instances except when a radicular cyst is responsible. Since roentgenograms indicate relative differences of density and not kind of pathology, a differential diagnosis based on roentgenographic interpretation is subject to error. This sometimes gives rise to some uncertainty in treatment planning. However, this is usually not as serious as might appear since clinical experience has shown that upon surgical intervention few such areas are cystic. In addition, study of many clinical cases has shown that the percentage of x-ray positive pulpless teeth which fail to heal following properly performed conservative root canal treatment is remarkably low (Figs. 1–8).

One recorded histologic study has shown that out of a total of 170 biopsies of radiolucent areas of pulpless teeth only 11 cases, or 6.4 per cent, were cystic; 143, or 84.9 per cent, were granulomatous; and 16, or 9.2 per cent, were diagnosed as periapical abscess, osteomyelitis (rare), and osteitis.[8]

In view of the foregoing it seems rational, except in unusual instances, to recommend routinely that periapically involved pulpless
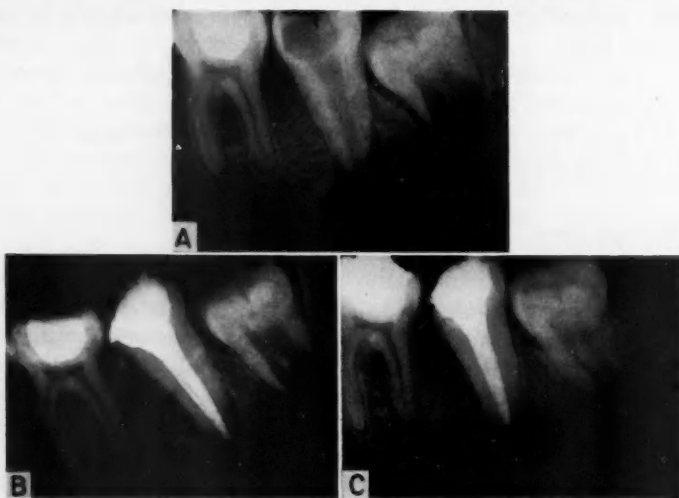
Fig. 1. Lower molar with gangrenous pulp and circumscribed periapical radiolucency. *A*, Before treatment; *B*, immediately after treatment; *C*, 14 months after treatment.
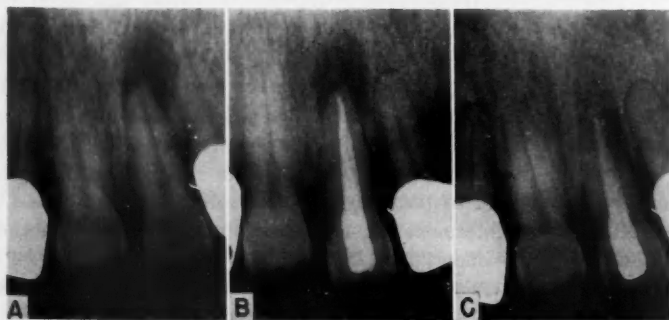
Fig. 2. Upper central incisor with gangrenous pulp and rarefied periapical area. A sinus tract which drained to the labial was also present. *A*, Before treatment; *B*, immediately after treatment; *C*, 4 years after treatment.

teeth be first treated by conservative root canal therapy. Should follow-up roentgenograms fail to show evidence of osteogenesis, surgery can be undertaken at that time.

Although roentgenographic signs such as radiolucent areas which are sharply circumscribed or punched out and which surround the apices of pulpless teeth, or large and spherical periapical radiolucent areas surrounded by radiopaque margins, are suggestive of radicular cysts, they should not be accepted as conclusive proof.[1]

Clinical evidence is likewise not completely reliable in the diagnosis of radicular cysts. For example, a number of pathologic conditions other than radicular cysts can cause localized bulging of the jaws with parchment-like feel.[9]

Usually the most reliable method of identifying radicular cysts is by microscopic study of tissue specimens. Even this method is not always conclusive, since in the early stages before full differentiation has occurred the criteria for identifying cysts are not clear-cut.

In instances in which the location of the tooth is favorable it may be possible to remove radicular cysts by the root resection technique, without extracting the involved tooth. The root canal must also, of course, be treated and filled.

Limited Accessibility

Two requisites for successful root canal therapy are to remove all debris inside the canal and to obliterate the canal space with a stable, non-irritating filling material. Treatment is therefore contraindicated in teeth with root canals containing necrotic debris which cannot be removed. Foreign matter left in the canal often causes failure for the following reasons: (1) It is infected or may become infected later; (2) it can prevent medication from disinfecting the canal; (3) it may decompose with the liberation of irritating products; and (4) it may interfere with hermetic sealing of the canal.

In carefully selected cases, if the tooth is favorably located and if the uncleansed portion of the canal lies in the apical portion of the root, surgical section of the root end with treatment of the remaining canal may succeed.

Teeth in which the gingival crevice is connected with the periapical region by means of a fistulous tract should be extracted. Root canal treatment fails because the periapical region is repeatedly infected by way of the sinus tract, which refuses to close.

Perforations

Teeth with a perforation of the root below the epithelial attachment produced during the operation generally fail to respond to root canal treatment. In such cases infection, tenderness and mobility commonly occur. Periodontal injury, infection and the difficulty of drying and filling the artificial tract contribute to failure. Treatment may succeed if the perforation is very small, such as that made with a root canal reamer; if the periodontal tissue is not seriously injured; and if the perforation occurs in the apical portion of the root. Treatment in such

instances is successful probably because the perforation lies distant from a common source of infection, the gingival crevice, and because the artificial tract is small enough to be plugged by a blood clot which later organizes. Another possible explanation is that root canal cement may be forced into such tracts and may seal them.

Root canal treatment of teeth in which the side of the root has been perforated by pathologic root resorption is contraindicated. There is little likelihood that the canals of such teeth can be cleansed or that the periodontal tissues will not be severely injured during treatment.

Incomplete Root Development and Non-vital Pulps

Teeth with grossly incomplete root development and non-vital pulps cannot as a rule be treated successfully by root canal treatment alone. These canals are large and funnel-shaped at the apex and cannot be thoroughly cleansed, disinfected, dried, and completely filled. In favorable cases it may be possible to save the tooth if the end of the root is first exposed by means of a surgical window, and if the canal is treated and filled while both ends are accessible.

CONDITIONS INDICATING QUESTIONABLE PROGNOSIS

There is general agreement that local conditions listed in the preceding paragraphs contraindicate conservative root canal therapy. However, a difference of opinion exists in reference to several other local conditions which supposedly also contraindicate root canal treatment. At least part of this disagreement stems from the fact that positive answers of "yes" or "no" in regard to prognosis cannot always be given. Variables may alter situations and give rise to results which differ. Nevertheless, conditions which sometimes prevent root canal therapy from being successful should not be listed as contraindicating treatment.

Root canal treatment is not necessarily contraindicated in teeth with the following conditions:

1. Teeth with a history of an acute dento-alveolar abscess.
2. Teeth with root apices which appear eroded in the roentgenogram.
3. Teeth with non-vital pulps which show extensive bone loss involving one-third or more of the root surface.
4. Teeth previously treated which have failed to respond to treatment, either by the appearance of acute infection or by periapical rarefaction.
5. Teeth so badly broken down that they cannot be isolated with the rubber dam during treatment.

The point of view in regard to the preceding five conditions which follows is based on the author's knowledge and clinical experience.

Acute Abscess

The acute dento-alveolar abscess runs a severe course and usually produces alarming symptoms. It is common knowledge that with the drainage of pus and tissue exudate, pain and swelling soon subside and the tooth once again becomes firm in its alveolus.

Teeth which have caused acute dento-alveolar abscesses usually respond favorably to root canal treatment. Clinical experience has shown that acute periapical inflammatory conditions as a rule have a more favorable prognosis than those of a chronic proliferative nature.

Eroded Apices

The belief that root canal treatment is contraindicated in teeth which roentgenographically show loss of bone and erosion of the root apex has arisen from the assumption that the root ends of these teeth are denuded of their periodontal membrane and that as a consequence the cementum is necrotic.

Histologic sections of jaws containing chronically involved pulpless teeth with periapical loss of bone commonly show localized root resorption.[1,2] It is not unusual for such resorptive processes to penetrate deeply into the root and leave eroded or crater-like zones of exposed dentin.

Root ends of pulpless teeth which according to clinical criteria have been successfully treated by root canal therapy have been sectioned and studied histologically. Under the mircroscope one sees demarcated zones of more recently formed cementum. Coolidge and Kesel[3] have presented evidence that such areas mark formerly active sites of root resorption. It seems reasonable to assume that the resorbed areas were rebuilt with secondary cementum after the environment in the periapical region was made favorable by root canal treatment. There is little doubt that this represents biologic healing of a hard tissue. Cementum and dentin as well as bone can therefore undergo repair and heal.

Roentgenographic evidence of root end erosion is suggestive of a vigorous periapical inflammatory response of perhaps long duration in which resorptive elements are unusually active. Infection, chemical irritants, and trauma are undoubtedly the causative factors most often responsible. If root canal treatment succeeds in eliminating the cause of injury, biologic healing of the hard tissues as well as of the soft tissue takes place.

Clinical experience backed by roentgenographic follow-up studies has clearly demonstrated that pulpless teeth with rarefied periapical areas and root resorption can heal completely following conservative root canal treatment (Fig. 3).

Fig. 3. Lower molar with gangrenous pulp and bone involvement of entire distal root. *A*, Before treatment; *B*, 2 years after treatment; *C*, 2½ years after treatment; *D*, 4 years after treatment.

Large Bone Loss

Some maintain that root canal treatment is contraindicated in pulpless teeth when loss of periapical bone involves more than one-third of the length of the root. Clinical results demonstrate that this contention has little justification. Periapical tissues do not lose their capacity to heal because of the size of bone involvement. Complete healing can occur even in cases where the loss of bone extends from the periapical region to the very crest of the alveolar ridge.

Conservative root canal therapy is indicated for treating pulpless teeth with unusually large rarefied periapical areas, unless a radicular cyst is present. Surgical curettage of such large periapical areas may devitalize adjacent pulps and create permanent bone defects.

A good rule to follow is that in all instances in which the diagnosis of a radicular cyst is not definite, and in which it is desirable to retain the tooth, carry out conservative root canal treatment and observe

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the results. If follow-up roentgenograms fail to show evidence of bone regeneration, surgical procedures can then be instituted.

The above plan of treatment is justified on the basis of clinical experience which has demonstrated that in most instances the patient saves the tooth and is spared unnecessary surgical trauma (Figs. 4
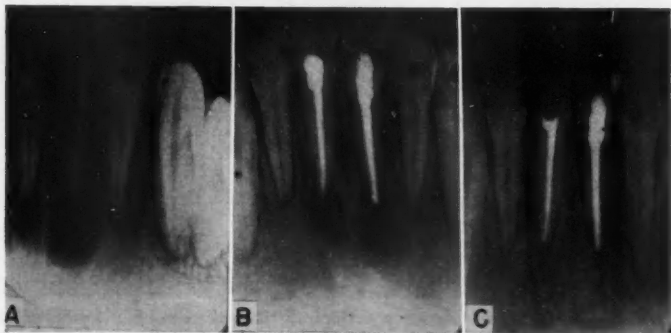
Fig. 4. Two mandibular incisors with putrescent pulps and rarefied periapical areas. Pulp cavities of both teeth contained thick, foul-smelling pus. A, Before treatment; B, immediately after treatment; C, 3½ years after treatment.
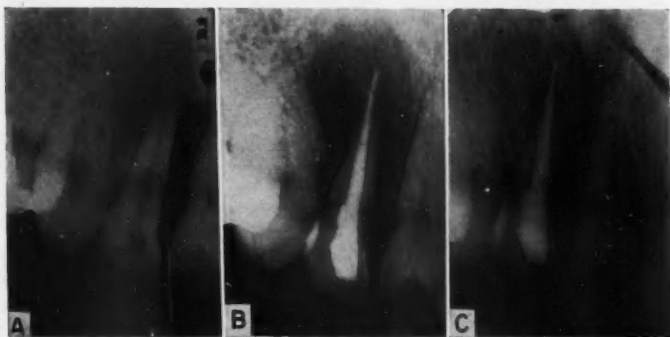
Fig. 5. Incisor with gangrenous pulp showing rarefied bone involving about one-half the length of the root surface. A, Before treatment; B, immediately after treatment; C, 1 year after treatment.

and 5). Should surgery prove necessary, postponement for a few months should not prove serious.

Failure to Respond to Previous Treatment

It is not necessarily true that previously treated teeth which failed to respond to root canal therapy should be extracted. There are many

reasons for failure in root canal therapy, the most common being poor and inadequate treatment. A high percentage of teeth in which root canal treatment has failed can be saved by carefully conducted re-treatment and filling of the canal (Figs. 6 and 7).
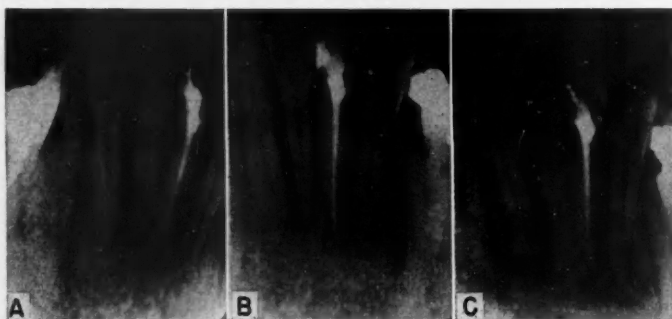
Fig. 6. Lower incisor showing periapical rarefied area, the result of unsuccessful root canal filling 3 years previously. A, Before re-treatment; B, immediately after re-treatment; C, 14 months after re-treatment.
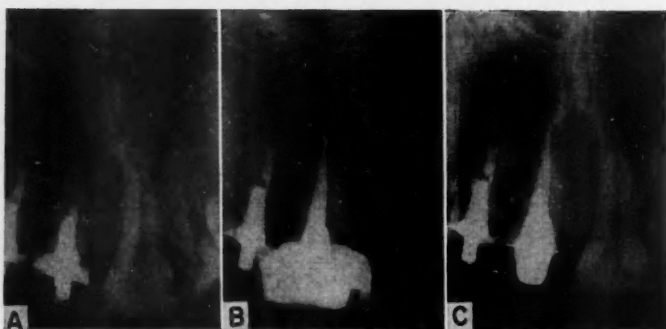
Fig. 7. Maxillary incisor showing periapical bone involvement. Note poor root filling and the presence of a metal post. A, Before re-treatment; B, after re-treatment; C, 10 years after treatment.

Inability to Isolate Teeth with Rubber Dam

Teeth which cannot be isolated by rubber dam because of extensive caries or fracture of the crown are difficult to treat because saliva which enters during treatment often re-infects the canal (Fig. 7).

In many instances, especially with maxillary teeth, it is possible for the operator to isolate the operative field with cotton rolls and gauze packs and thus prevent contamination. It is more difficult but not impossible to isolate mandibular teeth in this manner. Should

bacteriologic cultures repeatedly show growth, the prognosis becomes more doubtful. However, even in the latter instance, if the canal is well filled, it is possible for root canal therapy to be successful. Although the importance of negative bacteriologic cultures cannot be minimized it is common knowledge that many teeth have been successfully treated without culturing. Undoubtedly, some of these teeth had canals which were not sterile at the time of filling. Periapical tissues sometimes withstand considerable abuse and still respond favorably.

Systemic Influences

Root canal treatment is generally contraindicated in patients with certain serious constitutional diseases because their tissue resistance and capacity to heal are considerably below normal. Root canal treatment—or for that matter, surgery of any kind—involves a risk to these patients. Nevertheless, probably short of death, tissues never totally lose their potential to heal.

The active or uncontrolled phase of such diseases as diabetes mellitus, tuberculosis, the blood dyscrasias, and serious infectious diseases contraindicate root canal treatment. Generally the only kind of dental treatment permissible for these patients is that of an emergency nature. Should their disease be brought under control, root canal treatment may be undertaken.

Some question has been raised as to the advisability of root canal treatment for patients who have a history of rheumatic fever or congenital heart disease. Root canal treatment may be undertaken in these patients provided it is carefully done and provided surgical cleanliness is observed. If the tooth to be treated has a necrotic pulp or is grossly infected, antibiotic premedication of the patient is suggested to give added protection in the event of a transient bacteremia. The healing potential of the periapical tissues in these patients seems to justify treatment.

Although much has been written about the possible association of rheumatoid arthritis, fibrositis, bursitis, and neuritis with foci of infection, no positive proof has ever been presented.[4-7] As of the present there is no valid reason for denying root canal treatment to these patients.

Advanced age is sometimes listed as a contraindication for root canal treatment. Actually, elderly patients respond remarkably well to root canal treatment (Figs. 8 and 9). In many instances it is simpler to treat teeth in these patients than it is in the young because accessory canals in their teeth have usually calcified and the apical foramina are narrow.
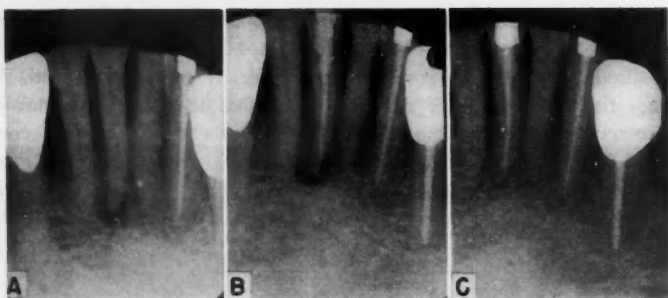
Fig. 8. Lower incisor in 66 year old patient showing periapical bone involvement. Patient presented with symptoms of an acute dento-alveolar abscess. A, Before treatment; B, immediately after treatment; C, 2½ years after treatment.
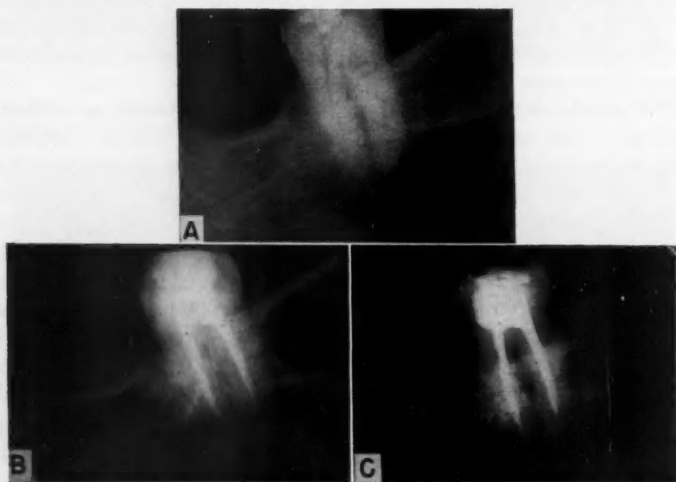
Fig. 9. Lower molar in a 73 year old patient which was exposed by buccal caries; the pulp was vital. A, Before treatment; B, immediately after treatment; C, 4 years after treatment.

Rarefied periapical areas seem to heal more slowly following root canal treatment in aged patients. In view of the decreased rate of calcium metabolism, this is not surprising. The important fact is that in the end healing will be complete.

CASES IN WHICH ROOT CANAL TREATMENT IS INADVISABLE

In an effort to cover the subject of this paper fully a number of examples may be cited in which root canal treatment is inadvisable

rather than contraindicated. In these cases successful root canal therapy is possible but inadvisable for various reasons. The following are examples belonging in this category:

1. When a prosthetic appliance is to be constructed and the tooth in need of root canal treatment is not necessary for the success of the appliance and its space can be included in the prosthesis.

2. When the patient is either unwilling or unable to receive adequate root canal treatment.

3. When the tooth has lost most of its supporting tissues owing to periodontal disease.

4. When the tooth in need of root canal treatment has no functional or esthetic value.

5. When a posterior tooth is so badly broken down that little or no sound tooth structure is left for a coronal restoration.

SUMMARY

1. Correct diagnosis is essential in the selection of cases for root canal therapy. Although pulp-involved teeth which are valuable may be saved by root canal therapy, this form of treatment is not always possible.

2. The prognosis in root canal therapy is often influenced by variables introduced by both patient and dentist.

3. Teeth can be divided from the standpoint of ease of treatment into two groups: (a) those relatively simple to treat and (b) those difficult to treat. The degree of difficulty which will be encountered often varies with the operator.

4. Local or systemic conditions which seriously interfere with the capacity of periapical tissues to defend themselves from injuring agents, or to heal, contraindicate root canal treatment.

5. Six conditions affecting the tooth are described which contraindicate root canal therapy. Root canal treatment coupled with root resection may succeed in some of these instances.

6. Root canal treatment often can be successfully carried out in (a) teeth with a history of an acute dento-alveolar abscess; (b) teeth with root apices which appear eroded in the roentgenogram; (c) teeth with non-vital pulps which show extensive bone loss involving one-third or more of the root surface; (d) teeth in which previous treatment has failed as evidenced by the appearance of acute infection or periapical rarefaction; (e) teeth so badly broken down that they cannot be isolated with the rubber dam during treatment.

7. Root canal treatment is contraindicated during the active or uncontrolled phase of certain serious constitutional diseases because

tissue resistance and the patient's capacity to heal are considerably below normal.

8. Certain precautions should be observed during root canal treatment in patients who have a history of rheumatic heart disease or congenital heart disease in order to give added protection in the event a transient bacteremia should occur.

9. Rheumatoid arthritis, closely allied collagen diseases and advanced age do not contraindicate root canal therapy.

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Diagnosis of Pain of Dental Origin

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In many dental schools, it appears that the subject of "toothache"—its possible causes, techniques of diagnosis and discussion of the pathology involved—has been neglected because the oral pathologist, the oral diagnostician, the endodontist and others have felt that some-one else was teaching the subject; so that only a smattering of in-formation has been available in a variety of textbooks and from a variety of clinical courses and instructors.

Considerably more investigation must be accomplished before we will have all the answers to the diagnosis of dental pain. Neverthe-less, it will be worth while to review our present knowledge of the variety of pathoses which may result in the production of pain in or around teeth, and the useful diagnostic tools and techniques which we have at hand at this time.

ETIOLOGIC FACTORS

Certainly the advance of *dental caries* through enamel to the ex-posure of dentinal tubules is the most common cause of the physiologic pain associated with the ingestion of salt or sugar which contacts the exposed dentin. Other factors in this realm include *erosion, abrasion, fracture, operative intervention* and *gingival recession,* any of which may expose dentinal tubules which become sensitive to these agents or to touch. Most of these processes may give rise to pulpal exposure with the entry of bacteria from the mouth into the pulp, contributing to a direct pulpitis with attendant symptoms. At the earliest exposure the major portion of the pulp may still be vital and the inflammation may be restricted to a tiny area in one exposed pulp horn, or it may spread throughout the pulp and ultimately cause its death.

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Operative exposure of the pulp is not as uncommon as we would like to believe (Fig. 1). Such exposure must occur frequently during the construction of crowns on teeth whose pulps have not receded because there has been no carious invasion to prompt secondary dentin formation. When such teeth are prepared under local anesthesia, no symptoms are produced at the time by the pulp exposure, and then the placement of anodyne preparations in the teeth often protects the pulp and masks symptoms which might indicate an exposure. Therefore, it is most necessary, especially when using local anesthesia,
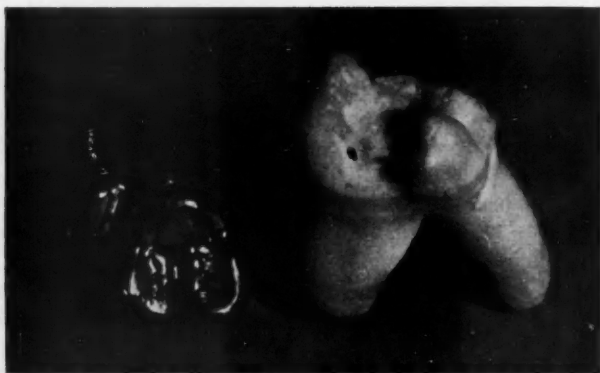
Fig. 1. This tooth was restored 4 months before it was extracted because of pain and a negative pulp test. The exposure (slightly enlarged) was apparent after removal of the restoration and cement. Many similar cases are found.

to inspect carefully the preparation for a possibility of exposure. The untreated, exposed adult pulp will practically always result in a poor prognosis for the tooth.

Acute trauma to a tooth may result in tenderness for a few days but under ideal circumstances the pulp may not lose its vitality. Under other conditions, the pulp may give a non-vital response owing to the trauma, and there may be symptoms of pulpitis and pain. It is not difficult to diagnose in this stage and the treatment is clear—either root canal therapy or extraction. On the other hand, no further symptoms may occur after the first few days of tenderness. After a long period of time, the tooth may turn dark, but before it does, sometimes routine roentgenograms reveal a radiolucency at the apex of this tooth. It is easy to assume that the pulp was strangulated during the acute trauma and died. However, what produced the radiolucency at the apex? One explanation of this situation is that the radiolucency at the apex is a granuloma (or possibly cyst) resulting from infection of the dead pulp. Whence came the organisms

causing the infection? The *anachoretic* effect has been described thus: When the pulp died, the remaining tissue within the pulp canal was without resistance to infection. There were no leukocytes carried in vessels because there were no living vessels present. There were no antibodies present; therefore, when the patient suffered a bacteremia, as we all do from time to time (transient bacteremias which may last perhaps 20 minutes at a time), the bacteria in the blood found their way to this area of low resistance, or no resistance, and multiplied there. The bacteria thus may have come from the blood stream from a distant portal of entry or they may have gained ingress into the lymphatics from inflamed gingivae and passed to the apical area where they localized.[3] This, then, will explain the roentgenogram of the apparently sound tooth with a radiolucency at the apex which has had no caries or external evidence of trauma. The pulp of course will not respond to the pulp test. This little point of differential diagnosis must be kept in mind when considering the periapical cementoma to be discussed later.

Differences in *inflammatory reaction to restorative materials* are apparent,[2,4,6] but the degrees of difference as yet have not been given their proper importance. It should be remembered that most restorative materials are placed in teeth which already have been attacked by caries and therefore, the teeth already have layers of secondary or irregular dentin, insulating the affected dental tubules from the pulp. This "insulation" may be a very effective barrier against irritative effects of restorative materials commonly used today.

<div align="center">

ABNORMALITIES OF TOOTH, PULP, OR
PERIAPICAL TISSUES WHICH MAY CAUSE PAIN

</div>

Exposed Dentinal Tubules

Patients may note pain and hypersensitivity when they place a fingernail or toothbrush on the cervix of a tooth, presumably because they are touching exposed dentinal tubules. Cementum has no demonstrable nerve supply so we can only assume in such cases that after some gingival recession has occurred, the cementum has been worn off the neck of the tooth. A comparable response occasionally may be elicited by touching fresh-cut dentinal tubuli, or those tubuli exposed by caries and other causes. This is the same response that is elicited after the ingestion of sugar or salt. When these crystals contact the dentin, it is theorized that a differential in the osmotic pressure at the external and pulpal ends of the dentinal tubuli is established,

thus causing pain. This is a physiologic response associated with a vital, usually normal pulp and requires attention only in determining whether this is the "toothache" about which the patient complains.

Thermal Changes after Recent Restoration

A patient may complain of toothache if he has recently had a restoration placed. This hypersensitivity to either hot or cold, or both, is often present for perhaps six weeks following the operation. In such cases, the pain is induced only by temperature change and disappears immediately thereafter. This has been called "active hyperemia" and since the discomfort tends to depart in time, it too has been considered physiologic rather than a pathologic response. More recent work indicates that there may be an actual pulpal inflammation, at least at the odontoblastic ends of the cut dentinal tubuli. If the symptoms persist for many weeks, or if they increase to become intolerable to the patient, then further steps must be taken to rule out true severe pulpitis. Relief for the patient with a postoperative "hyperemia" may be obtained by removing the restoration, examining carefully for the possibility of pulp exposure and, if none is found, placing a sedative temporary filling of zinc oxide and eugenol for a period of a few weeks and then restoring the tooth.

A "high" restoration of course may result in tenderness to percussion and should be reduced by marking and grinding.

Pulpitis

Most cases of pulpitis arise from entry of bacteria through one or more dentinal tubuli, and the primary site of the inflammation of the pulp is at the end of these tubules. If the pulp is unable to destroy the invading bacteria, then inflammation is likely to spread over more of the coronal portion of the pulp and subsequently along one or more of the root canals to the periapical area. No one can say how long it takes for a microscopic abscess at the end of a few dentinal tubules to become a diffuse pulpitis or periapicitis. No single diagnostic technique can accurately tell the operator the degree of the pathosis (see Figs. 2 and 3). As long as vital tissue is present within the pulp, a vital response may be gained from a variety of tests.

Spontaneous pain at night which prevents sleep is a sign of pulpitis. When a pulp test is applied to the suspected tooth, the patient may complain that the toothache has been induced. The persistence of

Fig. 2.   Fig. 3.

Fig. 2. This vital tooth was removed because of an extremely deep cavity and intermittent pain. Pulp exposure was expected. Serial microscopic sections did not reveal an exposure. This normal pulp could have survived with careful operative intervention. Compare to Figure 3.

Fig. 3. This vital tooth had troubled the patient for 2 years. A thin layer of secondary dentin exists under the cavity, but that portion of the pulp is necrotic (N). Vital tissue in the remainder of the pulp (V) seems to have laid down a central mass of metaplastic dentin. Compare to Figure 2. These two cases highlight the problem of determining clinically the status of the pulp— note similarity of symptoms.

pain long after a test source of heat has been removed seems to indicate a true pulpitis. Toothache at altitude (aerodontalgia) most commonly is due to pulpitis. Most often the vital aching tooth is not tender to percussion, but if it is, it must be assumed that there is still some vital tissue within the tooth and yet the inflammation has spread to the apex and involved the periodontal membrane.

The most common treatment for a true pulpitis is root canal therapy or extraction of the tooth. The placement of zinc oxide and eugenol, or calcium hydroxide, over a known infected pulp is not very effective, and the prognosis definitely is poor. The pain of the pulpitis may be relieved by such a procedure, but this only masks the fact that inflammation progresses anyway, with pulp death and subsequent periapical infection. Lack of follow-up on the part of the operator may give him a sense of security that is unwarranted, because the pulp death may occur without symptoms and the operator can detect this fact *only* by pulp tests. Capping of the young, incompletely de-

veloped tooth, especially when the exposure is not associated with carious invasion, is a different matter not to be considered here.

Periapical Pathosis

Granuloma. The dental granuloma most often is a direct result of penetration of decay with subsequent infection and pulp death. The granuloma itself is comparable to the "proud flesh" seen around an open wound on the surface of the body, which consists of hyperplastic fibrous connective tissue, increased numbers of swollen blood vessels and chronic inflammatory cells. Owing to the location of the dental granuloma, there also may be found in this tissue tiny islands of epithelium from the epithelial rests of Malassez.

Let us consider the development of a granuloma. A pulp dies as a result of infection. The bacteria and toxins penetrate to the vital, vascular tissues at the apex which have a much better collateral circulation than did the tiny string of the dental pulp. The periodontal tissue sets up an immediate reaction to the inflammatory agents; this takes place in a tightly bound area, the periodontal membrane space. With expansion of this small amount of soft tissue against tooth and bone, room must be gained. Therefore, the connective tissue at the apex, which is vital, is stimulated to form special giant cells or osteoclasts whose business it is to resorb bone. The plate of bone of the dental socket (lamina dura) is soon resorbed by the osteoclasts and the proliferating connective tissue of the dental granuloma fills the space formerly occupied by bone. We now have a bulbous mass of soft tissue where once there was bone; hence, the area is radiolucent.

The granuloma, then, is a living, inflamed fibrous wall around the apex of the tooth which is thrown up in an effort to contain the invading forces of bacteria and their toxins within the pulp canal. In most instances, there are no symptoms arising from the rather dormant and slowly forming granuloma. However, in the event of trauma to the tooth or debilitation of the patient, or an increased virulence of the organisms for some reason, an acute abscess may be formed with pus retained within the root canal; the attendant symptoms are extreme tenderness and possibly the formation of a sinus tract and/or swelling. The dormant or asymptomatic dental granuloma is a liability, and the involved tooth must either be treated or extracted.

Abscess. An abscess is a connective tissue sac filled with pus. Pus is a mass of dead and dying leukocytes in tissue fluid. Clinically, an abscess may be diagnosed only by the demonstration of pus such as that emerging from an opened pulp chamber or that released by

lancing. The pus is usually under pressure, which accounts for the acute tenderness. Microscopically, there is very little difference between a granuloma which is attempting to contain irritants within the pulp of a tooth and an abscess which is attempting to contain irritants within a tooth and perhaps within an area of unspecified size around the apex of the tooth. The basic treatment for an acute abscess, of course, is establishment of drainage, either by opening into the pulp chamber or by incising or extracting.

Periapical Cyst. The periapical cyst, sometimes called radicular cyst, periodontal cyst, or dental root cyst, always stems from a tooth with a non-vital, and therefore usually infected, pulp. A cyst is a cavity filled with fluid, the wall of which has an inner lining of epithelium and an outer capsule of fibrous connective tissue. The central cavity of the cyst may or may not be continuous with the cavity of the former pulp canal. The epithelial lining is believed to come from the epithelial rests of Malassez. In the presence of inflammation, epithelium tends to proliferate and may form an inner lining of a cavity.

A cyst may become acutely abscessed and the cystic lining may be destroyed in such case. An acute abscess may become chronic and thus undifferentiated from a granuloma; and either the granuloma or the abscess may become a cyst with proliferation of the epithelium mentioned above. There is a tendency to believe that a sharp, white "lamina dura" around a radiolucent area is indicative of the presence of a cyst. Many studies in which roentgenographic interpretation has been matched against microscopic analysis of the lesions indicate that this clinical interpretation is not reliable.[1]

Periapical Fibroma. The periapical fibroma, sometimes called ossifying periapical fibroma, cementoma, and other terms, usually shows up first as a radiolucent area because it consists mostly of fibrous connective tissue at this stage. It may later become partially ossified so that there may be radiopacity surrounded by radiolucency (because a bone-like substance which resembles cementum has been laid down in the center and is still surrounded by a fibrous connective tissue capsule). Later, the mass may be entirely radiopaque because it is now completely bony or cementoid (Fig. 4). This is not a true neoplasm. It occurs most commonly at the apices of the lower anterior teeth, but may appear singly or in numbers throughout a mouth in any region. It appears to be more common in females than in males. It is invariably associated with a vital tooth. If a negative vitality test is obtained, then one must assume that the radiolucency at the apex is due to inflammation. In the final diagnosis the only way this can be determined is by microscopic examination of the tissue. However,
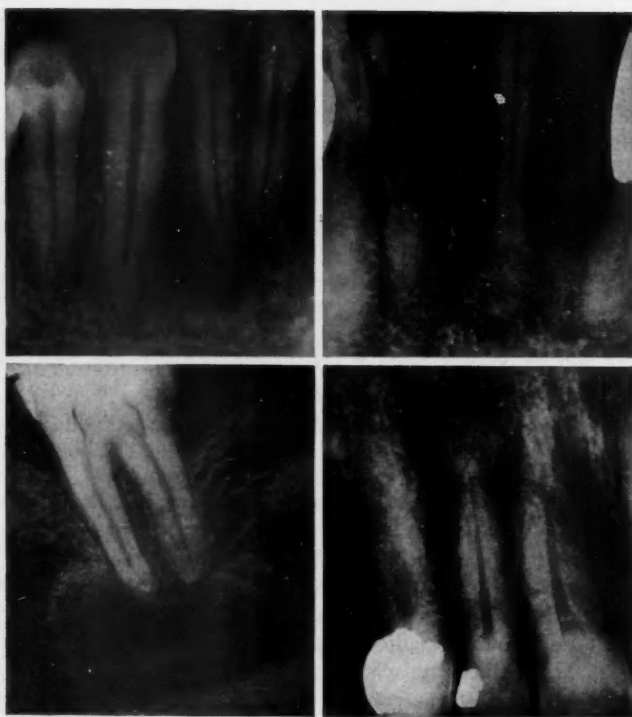
Fig. 4. The periapical abnormalities of these four cases are due to cementomas in different development stages. The teeth are vital.

many cementomas have been left in the mouth and observed for years without increase in size or development of symptoms.

DIAGNOSTIC TECHNIQUE AND TOOLS

History and Symptoms

The history and symptoms should be carefully recorded on the patient's record. Check the time of onset of symptoms, their duration and severity. Describe the nature of the symptoms, what seems to bring them on, previous history of trauma or operative care, tenderness during mastication, spontaneous pain at night or during meals. This information usually will indicate whether the pain is due to simple exposure of dentin or exposure of the pulp with attendant pulpitis or to other forms of pathosis.

Tooth Condition or Appearance

Obvious fracture of crown or root (Fig. 5) or discoloration of the tooth, or the presence of caries or a restoration all may be indicators of possible pulp pathosis. Carious exposure should not be judged by the appearance in the roentgenogram alone. Exploration or excavation of the cavity to reveal the presence or absence of pulpal exposure often is necessary. Tenderness to percussion, carefully evaluated and

Fig. 5. A tooth fractured *in situ*, with subsequent pulp death and periapical pathosis.

compared from tooth to tooth, indicates inflammation of periodontal membrane and most often is a sequel to pulp inflammation or death.

Roentgenography

Obliteration of the canal may possibly indicate that the tooth has suffered some onslaught in the past, particularly if it occurs in a young person in a single tooth in comparison to surrounding teeth. The depth of a restoration or a cavity may give some indication of whether or not it is the offender. The presence or absence of apical radiolucency certainly will help to determine whether this is the offending tooth. Taken with other symptoms, the presence or absence of thickening of the periodontal membrane at the apex may be indicative of trouble. Internal or external resorption of the tooth may be seen.

Pulp Tests

There are two major reasons for using pulp tests: (1) a positive test usually indicates that there is some vital tissue present to carry

the stimulus, and (2) the use of a pulp test may induce the pain that the patient has been complaining of, and thus point out the offending tooth. It is difficult to assign a great deal of importance to the graduated readings on the rheostat of most electrical pulp testers. Certainly, extreme variations in either direction must be considered as possible indicators of pathosis. Heat or cold may induce symptoms which may be helpful. The use of a bur or spoon during exploration or excavation of a cavity also is an effective pulp test. If no pain is encountered during this operation, suspect that the tooth is non-vital and determine vitality with other tests. This is one reason why it may be better to start operative procedures on a tooth without anesthesia.

Electrical Pulp Test. It is important to follow the directions furnished by the manufacturers of the various electrical pulp testers, whether they be of the bi-polar or uni-polar type. One of the newer models furnishes a high frequency current passing through a vacuum to a single pole and is called a uni-polar pulp tester.* It is not necessary for a second electrode to be in contact with the patient; however, the operator should contact the patient's skin or the response will vary. If the individual operator will faithfully and frequently use the pulp tester, he will find it a reliable instrument. The pole of the tester is usually moistened and placed on the dried tooth in question. If present, a control tooth in the opposite side of the arch is always tested. It is usually advisable first to check the pulp tester on the skin just behind the operator's fingernail to see if it is working satisfactorily. The point of the pulp tester should be placed on sound enamel over sound dentin whenever possible. Interpretation of a test which goes through a metallic restoration is difficult because the current may be carried to the gingiva if a proximal surface is present on the restoration. Cement under an inlay will carry this stimulus.[5]

A tooth with a "partial pulpitis" but with vital tissue remaining in some portions of the pulp will give a positive response to the test. A tooth with pulpitis which extends through one or more root canals and involves periapical tissue may at the same time give a positive response to a pulp test and be tender to percussion; although, most commonly, the tooth which is very tender to percussion will give a negative response to the pulp test. Seldom does a tooth with a radiolucent area at the apex give a positive response (except in the case of the cementoma). Sometimes a vital response may be obtained because of the conduction of the electric current to the periapical tissue by accumulated fluid in the root canal.

The electrical pulp test is not a completely reliable adjunct to diag-

* Pulp Vitality Tester, Type I and III Units, Ritter Company.

nosis, but it is one of the best that we have to use—without it, diagnosis of pulpal disease is very empirical.

Ice. The use of a piece of ice, which may be shaped in a little preforming cone in the refrigerator, often will help to determine which is the offending tooth. The non-vital tooth, of course, will give no response. The tooth with pulpitis may give an extremely early response to ice which is relieved upon removal of the irritant, or the ice may induce pain which persists long after the ice is removed from the tooth. Again, test the reaction of neighboring control teeth, and if results are inconclusive, resort to the use of heat and electricity.

Heat. Hot gutta percha flamed to the point of softness and applied to the tooth until pain is induced is an effective pulp test. The non-vital tooth usually will give no response whatever. The heat may induce and prolong pain simulating the toothache which the patient has complained of in the past. Hot water collected from the tap and applied to questionable teeth by means of a bulb syringe may stimulate a "toothache." Some operators have used an instrument, such as a wax spatula, heated in the flame for such purposes.

TABLE 1. *Response to Pulp Tests and Percussion in Pulpal and Periapical Pathosis*

PATHOSIS	RESPONSE TO PULP TESTS	RESPONSE TO PERCUSSION
Partial pulpitis	Positive. Possibly hyper- or hyposensitive	Negative
Diffuse pulpitis involving apical tissues	Positive. May vary over different areas of the crown, especially of multi-rooted teeth	Positive
Necrotic (non-vital) pulp	Negative	Positive or negative
Acute periapical abscess	Negative	Positive
Chronic granuloma or cyst	Negative	Negative
Periapical fibroma (cementoma)	Positive	Negative

Anesthesia

Well placed local anesthesia may help one decide which tooth is the offender. Given teeth with no obvious external signs of damage, vital response, and still subjective toothache, it sometimes is necessary first to determine which jaw may harbor the offender. In such cases a mandibular block may narrow the search to the upper or lower jaw. Well placed local infiltration may help one to determine which tooth

is the actual offender. This is not often required, but occasionally has been very useful in difficult cases.

Waiting Period

If all other means of diagnosis have failed to designate the offending tooth, it may be wise to use sedation and wait 24 to 48 hours. After that time the offending tooth may be tender to percussion. Pulp test results may change in a matter of hours, or certainly days, depending upon the progress of pathosis within the pulp chamber. A negative test after a period of waiting will certainly be an indication that the offender has been found. Patients who know that you have exhausted your ideas and are unable to determine which tooth to treat or remove are usually most willing to cooperate and undergo this painful waiting period. The use of the other alternatives, initiating root canal therapy or extracting a suspected tooth, too often has meant treating or removing a tooth with a normal pulp.

Postextraction Investigation

After removal of the suspected offender, examine it carefully for root resorption, fracture, attached granulation tissue and other signs of pathosis. Remove restorations and excavate cavities to see if there are exposures under them. Break open teeth to see if the pulp chamber is filled with pus.

* * * * *

Space does not permit discussion of such conditions, but the pain of maxillary sinusitis, pericoronitis, severe gingivitis, lateral abscess, and other afflictions may be called "toothache" by some patients.

SUMMARY

The judicious use of a careful history, observation, exploration and excavation of suspected teeth, roentgenograms, percussion and pulp tests, local anesthesia, careful reflection on findings, and sometimes waiting for more definite symptoms to develop usually will determine the cause of pain of pulpal or periapical origin.

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Pulp Capping and Pulp Amputation

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Need for Vital Pulp Therapy

Thousands of teeth are extracted annually in young adults and children as a result of deep carious penetration with potential or small pulp exposures. If the pulp is severely injured or dead, it should be removed and the root canals should be treated and filled. Unfortunately, many patients reject endodontic procedures as too expensive (especially in the younger age group) or as too difficult (as in molar teeth), and prefer to have these teeth extracted as a "simpler" or a "cheaper" solution, ignoring the fact that prosthetic replacement later will be far more time consuming and expensive as well as esthetically less satisfactory.

Teeth with pulps which are still vital can be treated more easily by pulp capping or by pulp amputation than by total extirpation of the tissue and obturation of the canals. Vital pulp therapy is today a safe and reasonably successful procedure. Where indicated, it is less time consuming and therefore less expensive (but also less certain) than root canal procedures. Its greatest virtue is that it may preserve many teeth in young adults and children which would otherwise be extracted. It is well to recognize that *at the present status of our knowledge,* preservation of teeth with carious involvement of the pulp by careful root canal therapy and filling is a more certain procedure than vital pulp therapy and is the *method of choice whenever the reparative ability of the pulp is in doubt.* Indirect pulp ther-

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apy and pulpotomy procedures are intended primarily in those situations where standard root canal procedures have been rejected by patient or operator or are technically not possible (as in very thin or tortuous canals of molars). Vital pulp therapy is also indicated in young and only slightly injured pulps whose reparative potential is still high.

Biologic Basis for Vital Pulp Therapy

The fundamental basis for indirect pulp therapy and pulp amputation is biologic rather than technical. Success depends on the ability of the pulpal connective tissue to heal itself and to seal off the injured or amputated portion of the pulp by the formation of reparative secondary dentin. The medicaments used are less important than the ability of the operator to follow biologic principles, to employ a surgically clean, gentle technique with respect for living tissue, and to protect the wound against reinfection.

The attempt to utilize biologic principles and to invoke the aid of living cells to repair damaged tissue is not new in dentistry. It simply follows current trends in endodontics. A decade ago, periapical rarefactions were generally treated by surgical removal (apicoectomy) immediately after root canal therapy. Today, the trend is toward avoidance of apicoectomy and dependence upon the ability of periapical connective tissue to heal itself, once the toxic agents have been removed and the canal has been sealed against reinfection. Less caustic drugs and milder sterilizing agents are preferred. Aseptic procedures and respect for living tissue are the order, based on the recognition that living tissue can combat infection and does heal.

Healing Potential of the Pulp

The pulp is a young and embryonic type of connective tissue with a tremendous cell reserve, and enormous healing potential (Fig. 1). The old aphorism that "an inflamed pulp is a dead pulp" is not true, as attested by thousands of carious teeth preserved for years by indirect pulp therapy and pulp amputations (Fig. 2).

Limitations on Healing Potential. This young tissue is housed within hard and unyielding walls of calcified dentin. Its vascular supply is restricted by the small openings of the apical foramina. *This lessens but does not destroy* the ability of the pulp to repair itself. The potential is enormous; these limitations merely leave it great.

In order to heal, the pulp needs only a modest amount of help:

1. Removal of the noxious agents, particularly the bacteria in the overlying carious dentin.

Fig. 1.   Fig. 2.

Fig. 1. Photomicrograph showing the formation of reparative dentin under a deep carious lesion.

Fig. 2. Photomicrograph showing localization of pulpal inflammation 6 days after application of a sedative dressing (zinc oxide and eugenol).

2. Avoidance of excessive surgical trauma or chemical irritation (avoid caustic drugs or irritant fillings).

3. Protection against reinfection. Marginal leakage around the filling over the amputated pulp and subsequent reinfection is the major cause of failures following pulpotomy procedures. In fact, marginal leakage is a basic cause of failure following all operative procedures.

The remarkable vitality of the pulp and its ability to repair injury are seen in various ways. Under chronic deep caries the entire pulp chamber often becomes filled in by reparative secondary dentin, if the caries is intermittent or arrested (Fig. 1). A period of rest also permits the pulpal cells to produce reparative dentin under very deep and injurious cavity preparations and even under noxious fillings such as silicate cements. Pulp capping and pulp amputations are often successful in spite of empirical methods which violate fundamental biologic principles. Closer attention to and use of biologic principles should result in more successful pulp amputations.

Differences Between Primary and Permanent Teeth in Pulpal Healing after Pulpotomy. A number of reports have appeared recently which suggest that pulpal healing following amputation occurs more frequently in young permanent teeth (Englander et al., 1956) than

in primary teeth (Via, 1954; Laws, 1957). Very few histologic studies have been pursued using primary teeth. MacDonald (1957) indicates that the defensive power of the deciduous pulp is very poor because localization of inflammation and infection is poor in contrast to the excellent localization of inflammation and infection in permanent teeth as shown by James et al. (1957) (Fig. 2). Clinically, bridging is more frequently seen and pulpotomies are, in general, more successful in permanent than in primary teeth.

Internal resorption is also observed much more commonly in primary teeth following pulpotomy (Rabinowitch, 1956). This is probably due to the tendency for the inflammation to be more diffuse in these teeth and because of the much higher resorption potential.

Indications for Vital Pulp Therapy

1. Fractured Incisors with Open Apical Foramina. Vital pulp therapy and vital pulp amputations are indicated whenever complete obturation of a wide canal with open apex would be difficult or impossible, as in a young fractured incisor or where marked discoloration of the tooth crown would be objectionable.

2. Primary Molars. Vital pulp amputations are indicated also when obturation of a thin, tortuous canal would be too difficult or impossible, as in primary molars (or third molars).

3. Young First Permanent Molars (6–9 Years) and Young Second Molars and Bicuspids (12–16 Years.) These teeth are more easily preserved following exposure by caries by means of vital pulp therapy than by complete endodontic procedures. The latter can be performed later if necessary.

Contraindications to Vital Pulp Therapy

1. Badly broken down crowns which cannot be restored without the use of a post.

2. If root canal therapy and obturation can be carried out simply and successfully.

3. Absence of healthy vital pulp tissue.

Note: Age of the patient is not considered a valid contraindication.

PULP CAPPING

Pulp capping is the placement of a therapeutic agent over a clinically visible or microscopic exposure of the pulp. As used today, the term usually refers to a procedure in which the pulp is not subjected to surgical amputation.

The purpose of pulp capping is to sterilize the infected, carious dentin, and if possible also the infected portion of the pulp itself, *without injury to the pulpal cells* and thus to enable the pulpal cells to wall off the injured area by a barrier of reparative secondary dentin (Fig. 3).

Principles. A pulp which has been recently injured and possibly infected by caries and is presently in a state of acute inflammation, is not a good candidate for surgical amputation. Nor should any tissue which is acutely inflamed and infected be subjected to surgical procedures, since it is likely to become necrotic as a result of such indiscretion.
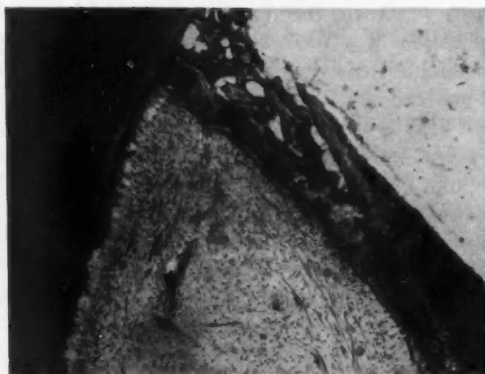
Fig. 3. Photomicrograph showing dentin bridging under a small exposure 61 days after applying a dressing of calcium hydroxide and sealing with zinc oxide and eugenol.

The infected and inflamed pulp should be sedated and disinfected prior to any surgical procedure, whether this be amputation or complete extirpation of the pulp. Leading endodontists (Blayney, Kesel, etc.) have for years taught that an anodyne-antiseptic dressing (usually eugenol) should be sealed into the chamber at the first sitting prior to vital pulp extirpation, to sedate and disinfect the pulp. Extirpation of the vital but infected pulp at the first sitting invites necrosis and infection of the pulpal remnants near the apex.

Clinical Application. In potential and in small pulp exposures, mechanical removal of the superficial layer of grossly infected necrotic dentin and sterilization of the remaining tissue often permits the underlying pulpal cells to lay down a barrier of reparative secondary dentin and thus to wall off the injured area with secondary dentin (Fig. 3). This often (in 40 to 60 per cent of all cases) eliminates the need for surgical amputation of the pulp later.

The *first visit* in the treatment of *all* deep carious lesions, with or without pulpal exposures (but with pulpal injury), should consist of (*a*) the removal of infected dentin and (*b*) sealing in of a sedative germicidal agent—*without leakage*—to permit recovery of the pulpal cells and initiation of the reparative process.

Clinical and experimental histologic evidence suggest that *indirect pulp capping* over small exposures is uniformly more successful than pulp amputation.

Direct vs. Indirect Pulp Capping

Direct capping involves the removal of all overlying infected dentin and application of a medicament *directly* to the exposed, nonbleeding (or minutely bleeding) pulp tissue so that the drug (usually a germicide and calcium salt) acts *directly* on the pulpal cells. Failures in pulpal healing usually occur because most drugs used are irritant to the pulpal cells and are therefore, at least potentially, injurious to the pulp.

A more satisfactory result is achieved when there is a thin, intervening layer of sterilized dentin between the germicide and the pulpal cells. This is referred to as *indirect capping*.

Because of the large numbers of clinical failures following direct capping with strong caustic germicides, this procedure is rarely used today. Indirect pulp therapy is the procedure of choice.

Drugs Used

Mild Germicides. Mild germicides should be used which are sufficiently strong to inhibit bacterial proliferation (bacteriostatic) or kill the organisms (bactericidal) but do so *without injury to the pulp*. It is not rational to pour phenol or caustic silver nitrate into an open wound in order to destroy the organisms therein if by so doing one also destroys the living cells upon which one depends for the reparative work.

Penetration of Germicide. It is important to realize that the brief wiping on of a germicide (such as phenol) for a period of 1 to 3 minutes does not permit penetration of the germicide into the substance of the dentin (sound or carious). The germicide therefore does not make contact with the organism. Because phenol requires a long time to diffuse into the dentin, it is ineffective in the sterilization of dentin. It is a *surface* disinfectant only. Since it does not reach the pulp, it also does not harm it. Silver nitrate penetrates quickly and deeply, but it may be injurious to the pulp when it does so. Also, it is not re-

markably effective in destroying the bacteria, so that it has not even this virtue to balance its potentially injurious effects on the pulp.

Time of Contact. There is a minimal time during which the germicide must be in contact with the organism in order to destroy it. Phenol (95 per cent) must be in contact with bacteria for a minimum of 6 minutes; boiling water must contact the organism for 15 minutes. The presence of extraneous organic matter (or dilution by water) greatly decreases the action of the germicide. For this reason, silver nitrate is not effective on living tissue because the protoplasm within the tubules precipitates the solution before it can contact the bacteria for a sufficient time to be effective.

Effective Sterilization of Dentin. Sterilization of carious dentin must be predicated upon the *sealing in of a non-irritatnt germicide for a period of days*. Direct evidence is still lacking but it is thought that eugenol requires 1 to 2 weeks and camphorated paramonochlorphenol requires 2 to 3 weeks for effective sterilization of infected dentin. These drugs are non-irritant to the pulp (Perreault et al., 1956). In this connection, the potentialities of the antibiotics as dentin-sterilizing agents should be given much more serious consideration by scientific investigations, than has been done to date. A few pilot studies indicate that a polyantibiotic mixture might well satisfy the criteria desired—high potency and rapid penetration with little or no damage to pulpal cells.

Sealing of Medicament. The potency of the germicide seems to be much less important than the integrity of the marginal seal.

Powerful medicament + leakage → acute death of pulp due to irritation and re-infection.
No medicament + perfect seal → slow death of bacteria (probably), no irritation, slow recovery of pulp.
Mild germicide + perfect seal → death of bacteria, no irritation, rapid recovery of pulp.

Perfect sealing of a cavity, over a long period of time, is not yet a realized objective in dentistry. Even an amalgam filling shows marginal leaking soon after insertion (Schoonover and Souder, 1951; Armstrong and Simon, 1951; Wainwright 1956). Zinc phosphate cement shows marginal leakage in a few days (Grossman, 1932). Gutta percha leaks within minutes. Pulpotomies covered only with zinc phosphate cement survive only for a short time. When covered with amalgam over the cement, they survive well for a year or two but succumb eventually to the ingress of bacteria and fluids along the margins. At this time, it seems that zinc oxide and eugenol is the best sealant presently available, although it is still far from adequate for this purpose, especially when used alone as a filling material

(Massler and Ostrovsky, 1954). *It is most effective when used as a sealant base under a strong filling material* like amalgam or inlay or shell crown.

The sealing of the cavity and therefore the sealing in of the medicament is more important and critical in vital pulp therapy than in root canal therapy because the wounded pulp tissue is much closer to the surface and more easily invaded by bacteria. An embryonic tissue like the pulp is an excellent culture medium for bacteria.

Conclusion: Use a *mild* germicide and a *good seal.* It is very important to the success of vital pulp therapy that (1) a stainless steel band be cemented into place before attempting vital pulp therapy to avoid the almost inevitable leakage at the proximal cervical margin under temporary fillings, and (2) a zinc oxide and eugenol sealant be used under the temporary or permanent filling material.

The great majority of failures after pulp capping are due to marginal leakage of the temporary filling!

A Technique of Indirect Pulp Capping

1. Apply a pinch band. Make sure *before* you begin that the temporary seal will *not* leak. It is not rational to attempt treatment if leakage or loss of filling will occur and undo all your labor.

2. Remove the superficial layer of bacteria-laden, grossly infected, necrotic dentin. This layer contains 90 per cent of the acidogenic and/or proteolytic bacteria involved in caries. Stop when you reach the layer of still vital and painful dentin. This layer is decalcified and stained but contains vital odontoblastic processes. Well meant but injudicious excavation at this time may further injure the already injured odontoblasts, pulp cells and blood vessels, causing hemorrhage into the pulp.

   a. If removal of the superficial layer of necrotic debris and dentin exposes (or almost exposes) the pulp tissue, place a protective layer of calcium hydroxide over the area to protect the pulp against the possible irritant action of the superimposed germicide. Since pure calcium hydroxide in water has a pH of 11.6, it is best to use this material in a diluted form such as calcium hydroxide in methyl cellulose (James et al., 1957).

3. Seal in the germicide. Any mild germicide may be used which is effective and non-irritant. Camphorated paramonochlorphenol, thymol and zinc oxide, even camphorated phenol may be used. The drug of choice at present, in our opinion, is zinc oxide and eugenol because (1) it is non-irritant and effective and (2) it is easily manipulated to meet different situations.

a. If removal of the necrotic dentin leaves a hard layer of clear dentin, the zinc oxide and eugenol can be mixed thick and hard. This will release a minimal amount of eugenol. The material can also be left *in situ* after it is set to serve as an insulating base and sealant under the permanent filling.

b. If it becomes necessary to leave some carious and infected dentin within the cavity because the child is uncooperative or the tooth very painful, the operator can place a *thin* mix of zinc oxide and eugenol. This will release a considerable amount of eugenol to sterilize the cavity contents. In extreme cases, a pellet of cotton saturated with eugenol may be placed for a day or two to relieve extreme pulpitis.

c. Cover the germicide with a layer of fast-setting zinc oxide–eugenol cement. Add 1 drop of a saturated solution of zinc acetate to accelerate the setting of this mixture or use any one of the many commercial zinc oxide–eugenol preparations which set rapidly and hard (periodontal packs; zinc oxide–eugenol impression pastes and temporary cements). These preparations make good sealing agents and excellent cement bases.

d. Cover with a zinc phosphate cement to prevent dislodgement of the medicaments. Add amalgam filings to the cement to increase its hardness, if indicated.

4. Allow 4 to 6 weeks for pulpal healing to take place. Do not be impatient. Remember that it takes approximately 2 weeks for new odontoblasts to differentiate and to replace the injured cells. Another 2 to 3 weeks is required for a thin layer of reparative dentin to be formed by these cells under the area of injured dentin.

If the cavity is opened too soon (in less than 10 days), the area may appear to be hypersensitive. This is due to the formation of new cells, capillaries and nerve endings and merely indicates that active healing is taking place.

If normal sensation is diminished, it may indicate pulpal degeneration, either partial or complete. It is best, even then, to leave the pulp alone lest secondary infection be introduced into the dying pulp with subsequent acute flare-up. *If the germicide is potent and the sealing remains intact, a sterile necrosis of the pulp results.* It is then easier and safer to do a complete pulpectomy, a thorough curettage and root canal filling in the usual fashion with a minimum of effort.

If the practitioner is uncertain as to the prognosis and course of pulpal healing, he should open the cavity in approximately 2 to 3 weeks under a rubber dam, carefully remove the remaining carious dentin and re-check the pulpal response directly. If vitality is still present, re-seal as before, for 4 weeks.

5. We have found that secondary reparative dentin forms under the

area of potential or small exposures in 80 per cent of the cases so treated in young permanent teeth (Fig. 3).

PULP AMPUTATION

Pulpotomy is the surgical amputation of the bulbous portion of the pulp within the pulp chamber. Partial pulpotomy is the removal of a part of the pulp within the pulp chamber. Total pulpotomy is the removal of the entire contents of the pulp chamber, to the entrance to the root canals.

Pulpectomy is the surgical amputation of the pulp within the root canal. Partial pulpectomy is the removal of part of the pulp tissue
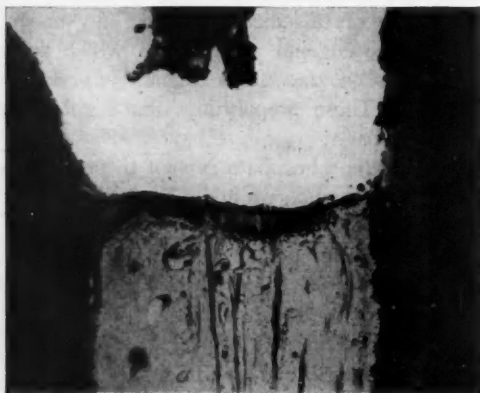
Fig. 4. Photomicrograph showing a dentin bridge forming 28 days after pulp amputation. The amputated pulp was covered with a dressing of calcium hydroxide and sealed with a fast-setting zinc oxide and eugenol cement.

within the canals. Total pulpectomy is the removal of the entire contents of the root canals to the apical dentino-cemental junction.

Since the introduction of new terminology, even when carefully defined, causes as much confusion as clarification, we prefer to refer to these procedures as "vital pulp amputations." This term is less subject to misunderstanding and lends itself to more exact definition.

Surgical Principles. Successful healing following pulp amputation depends on the ability of the pulpal cells to produce a reparative wall of calcified dentin (Fig. 4). This ability depends on (1) strict observance of surgical principles and (2) the postoperative care used to protect the exposure against reinfection. The medicament used over the wounded pulp may be helpful, but is not as critical as was formerly believed.

A Procedure for Pulp Amputation

First Visit—Sterilization of Dentin, Arrest of Decay and Sedation of the Pulp.

1. Remove the superficial debris and soft necrotic layer of infected dentin as gently as possible. Stop as soon as the patient feels definite pain.

2. Chip away all undercut enamel with a sharp chisel. Be careful not to hit the cavity floor. Remove all *peripheral* carious dentin with a sharp spoon by peeling it away from the underlying dentin. Stop only when the area becomes very painful, since this indicates injury to the odontoblasts.

Do not touch the carious dentin immediately over the exposure lest you push infected dentin into the pulp.

3. If no actual exposure can be seen clinically and a layer of carious dentin still remains over the vital pulp, try indirect pulp capping as outlined before.

4. If a small exposure is noted (usually non-bleeding), apply a thin layer of calcium hydroxide paste and cover with a thin mix of zinc oxide and eugenol. The layer of calcium salt will protect the pulp against the direct action of the eugenol. Cover as before with a thick mix of fast-setting zinc oxide and eugenol cement and zinc phosphate cement. Recall in 7 to 14 days for a partial or complete pulpotomy.

If, in the clinician's judgment, the pulp is relatively young and healthy, this dressing may remain undisturbed for 4 to 6 weeks as a pulp capping. In many instances, capping a small pulpal exposure with calcium hydroxide, plus indirect sterilization and sedation with zinc oxide and eugenol, permits a young and healthy pulp to recover fully. New odontoblasts differentiate and form secondary dentin to seal over the exposure. Complete closure of the exposure was found in 60 per cent of the teeth thus treated when the area was re-examined 4 to 6 weeks later.

5. If the pulp is obviously exposed, severely damaged and partially necrotic, remove all the grossly infected and necrotic portion of the pulp until you reach vital pulp tissue as evidenced by pain. Stop at this point. Try not to produce bleeding, since the site is grossly infected, but do remove as much of the necrotic dentin and pulp as possible.

Seal in eugenol on a paper point or cotton pellet. Cover with a thick mix of zinc oxide and eugenol cement and protect with zinc phosphate cement. Recall in 3 to 7 days for partial or complete pulpotomy.

Second Visit—Surgical Amputation of the Pulp.

1. Make a pinch band and cement it in place—especially if the tooth is badly broken down. A preformed stainless steel orthodontic band can also be used. Make sure the fit at the gingival level is perfect. The band should extend no higher than the level of the marginal ridge.

2. Inject procaine for anesthesia.

3. Arrange sterile tray of instruments.

4. Apply rubber dam.

5. Open and extend cavity to final outline form. Do not attempt to do the pulpotomy before the cavity outline is completed and the floor of the cavity is in open view.

6. Remove the carious dentin (now sterile) remaining over the exposure with a sterile No. 6 round bur. Clean this bur thoroughly or replace with a new bur before proceeding, to avoid pushing dentin spicules into the pulp. A very sharp spoon excavator may be used if the carious dentin will peel off in one piece.

7. When the exposure is clearly visible, amputate the pulp with a new, sharp No. 6 round bur or a sharpened spoon. Amputate to the level of the root canal(s). Work rapidly and amputate with one motion. Pain is minimal if you work quickly and with precision. Don't scratch.

8. Allow the pulp stump to bleed freely for at least 3 to 5 minutes. If bleeding does not occur or is very slow, amputate at a lower level. Do not use astringents. Sponge away the blood with sterile cotton pellets until bleeding stops.

9. When bleeding has stopped, cleanse the cavity preparation with warm sterile water.

10. If bleeding is excessive or prolonged, blow warm air over the blood to accelerate clotting.

11. Allow only a *thin* clot to form over the amputation. Sponge away all excess. Permit the clot to become firm. *Do not hurry.* Be sure you have a good firm clot before proceeding. Normal clotting time is approximately 3 to 5 minutes.

12. Place a thin layer of calcium hydroxide paste gently over the clot. If the clot dissolves and bleeding is renewed, you have been too impatient. Begin again.

13. Cover with a layer of zinc oxide and eugenol cement. Press the mixture against the lateral walls of the cavity and allow it to flow over the dry layer of calcium salts so as not to disturb the blood clot.

14. Cover with fast-setting zinc oxide and eugenol or zinc phosphate cement. Under-contour the filling lest it be cracked out and lost.

15. Remove rubber dam and test the occlusion using articulating paper. Remove enough cement to keep the tooth well out of occlusion. A fractured filling will undo all your work.

16. Dismiss the patient for 4 to 6 weeks. Recall the patient for re-examination of the site of amputation for evidence of healing, before placing the permanent restoration. After the first dozen pulpotomies are performed successfully, the operator may omit the recall and place the final restoration at this time.

Third Visit—Follow-up (After 4 to 6 Weeks).

1. Take two roentgenograms, one periapical and one bite-wing.

2. Apply rubber dam and arrange sterile tray.

3. Open the cavity carefully. Use a sharp No. 6 round bur to remove the hardened phosphate cement and zinc oxide and eugenol. Avoid pressure directly on the pulp. When the layer of calcium hydroxide is reached, wash carefully with warm saline until the amputated pulp stump is clearly visible. Remove all debris by washing with *warm* saline. Record which of the conditions listed below obtains:

a. A calcified bridge of dentin covers the amputated area.

b. A dense connective tissue covers the amputated area. The stump appears pink and healthy and is not bleeding.

c. A firmly adherent blood clot covers the area of amputation. Do not disturb.

d. A loose, non-adherent soft clot covers the amputation. This usually loosens and is lost during the cavity toilet. Bleeding may occur.

e. The pulp stump is covered with a serous exudate.

f. The pulp is covered with a drop of clear white pus.

g. The pulp appears to be degenerating and necrotizing.

4. In the majority of cases, the exposure is covered with a calcified bridge of dentin, indicating that healing has occurred. If the pulp stump is covered by connective tissue, healing is well under way. In either case, wash carefully with sterile saline. Dry. Place a thin layer of calcium hydroxide paste over the area and cover with fast-setting zinc oxide and eugenol cement. Allow to set and place the permanent restoration.

5. If the blood clot is still present and partially organized, the pulp stump is normal but healing is only begun. Do not disturb the clot. Re-cover the area with calcium hydroxide and zinc oxide and eugenol and re-examine in 4 to 6 weeks.

6. If a serous or purulent exudate is found, healing is retarded. A serous exudate indicates the presence of irritation and inflammation. Pus indicates that some break occurred in the sterile technique. In either case, wash carefully with a mild chlorine solution and gently

re-amputate at a slightly lower level. Proceed as during the second visit. Allow a clot to form, cover with a paste of calcium hydroxide and seal with zinc oxide and eugenol. Re-examine in 3 to 4 weeks.

7. If the amputated pulp appears necrotic or degenerating, the pulp was probably grossly infected during the amputation or severely injured by excessive pressure over the amputated stump. Wash carefully with a mild chlorine solution. Re-amputate at a lower, vital level and proceed as during the second visit.

Discussion. In general, amputation of the grossly infected pulp at the first visit and covering with an aqueous paste of calcium hydroxide, which is highly alkaline, will result in the formation of a wide layer of necrotized tissue which quickly becomes calcified. This type of bridging is clinically acceptable.

From the histopathologic point of view, better results are obtained if amputation is delayed until the pulp is sedated and is no longer grossly inflamed, and is sterile. Healing is more physiologic. If a thin blood clot is allowed to cover the wound and if a mildly alkaline calcium compound is used under a palliative germicidal dressing of zinc oxide and eugenol, the pulp is further encouraged to return to normal. Necrosis of the underlying pulp tissue is avoided and pathologic calcifications in the pulp do not occur. Instead, bridging occurs by the formation of regular tubular dentin. This takes place more slowly but is more physiologic. From the clinical point of view, the pulp and the patient are also more comfortable.

Evaluation of Success

Evaluation of the success of a series of pulpotomies poses a problem in the use of criteria. If preservation of the tooth is the only criterion, then pulpotomies have been reported as successful in up to 92 per cent of cases. However, as the clinician becomes more critical and demands more exacting standards, the percentage drops quickly. The clinical symptom of pain is rarely important, since sterile pulpal degeneration without pain is the rule rather than the exception. If roentgenographic examination after 1 year is added, the successes drop to about 75 per cent because symptomless periapical rarefactions appear under permanent teeth and internal resorptions in primary teeth. As the period of postoperative evaluation increases (2 to 5 years), the successes may drop to about 65 per cent, probably owing to marginal failure of the filling.

Hess and co-workers[4,5] properly consider histologic evaluation the best criterion of success in that pulpal healing can be observed directly and in detail. Such studies indicate that pulpotomy procedures

result in healing histologically in about 80 per cent of cases as compared to the same material clinically evaluated as 90 per cent successful.

It is obviously impossible to section and examine histologically all the pulpotomies done on a clinical basis. Since roentgenographic evidence and clinical symptoms are not always reliable, it is suggested that visual re-examination of the amputated pulp stump after 4 to 6 weeks would more exactly reveal the status of the pulp. The criteria for such evaluation have already been discussed.

SUMMARY AND CONCLUSIONS

The method of indirect pulp capping for the preservation of the pulp under very deep carious lesions with potential or small pulp exposures has been used successfully for many years in both adult and young teeth.[1,2,6,7] Partial pulpotomy and partial pulpectomy to preserve the vitality of the unaffected pulp tissue under large exposures have similarly proved very successful in preserving thousands of teeth which otherwise would have been extracted.[3,5] The only contraindications are extreme destruction of the crown, so that the tooth cannot be restored, and the absence of healthy vital pulp tissue.

The technique is not difficult or tedious. Pain is minimal, and the results are gratifying to both patient and doctor.

In deep cavities with potential or very small exposures, indirect pulp capping yields best results. The superficial layer of necrotic debris is carefully removed until vital dentin is reached. The area is then protected with a thin layer of calcium hydroxide paste and covered with a layer of zinc oxide and eugenol. This arrests the decay and permits the pulp to recover from the injury. New odontoblasts then differentiate and form a protective layer of secondary dentin under the lesion.

Direct pulp capping of small exposures (or small surgical amputations) by a layer of calcium compound (generally calcium hydroxide) aided by the mild germicidal action of a covering of zinc oxide and eugenol cement, also permits the pulp to heal and to seal off the exposure with secondary dentin.

When the pulp is grossly exposed and partially necrotic, amputation of the degenerating portion becomes necessary. This should be performed only after the acute inflammation has subsided and the pulp is sedated by sealing in zinc oxide and eugenol for approximately one week. After the amputation, a thin blood clot with a mildly alkaline calcium compound covered by a dressing of zinc oxide and eugenol promote physiologic healing.

Indirect pulp capping and vital pulpotomy permit the preservation and restoration of many teeth in children and adults which otherwise would be lost by extraction. Further research and improved techniques will surely expand the usefulness of this procedure in the near future.

Many of the pulp studies mentioned in this review were carried out at the Great Lakes Naval Training Center, Great Lakes, Illinois, under the direction of Lieutenant Commander Harold R. Englander. Photomicrographs by William M. Winn.

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Endodontic Instruments and Instrumentation

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BASIC INSTRUMENTS

The selection of instruments to be used in endodontic cavity preparation is related to the location of the preparation within the tooth.

Coronal and Intracoronal Preparations. Preparations upon and within the crown of the pulpless tooth are completed almost entirely with power-driven rotary instruments (Fig. 1A). For initial entrance through an *enamel* surface, the preferred instrument is the diamond point. These enamel penetrations are confined to the lingual surfaces of the anterior teeth and the occasional posterior tooth with a virgin occlusal surface. Initial entrance into posterior teeth is invariably made through a carious or restored surface, hence the carbide round bur is the instrument of choice.

Once the enamel has been penetrated, round carbide burs are used for dentin removal in both anterior and posterior teeth, whereas extension of the external cavity outline in the enamel is well accomplished with either a carbide fissure bur or a specially designed diamond point (Fig. 1A). Accelerated operating speeds with any of these instruments will increase patient comfort.

Intraradicular Preparation. The hand-powered specialized instruments for the mechanical preparation of the root canal are used only for endodontic therapy. Rarely is a power-driven instrument used in the root canal.

The two primary instruments for canal enlargement are the reamer and the file (Fig. 1B). Both reamers and files are supplied with long and short handles, long-handled instruments being used primarily in the maxillary anterior region and short-handled instruments in all posterior and mandibular anterior teeth. Reamers and files are available in sizes 1 through 12; though the problem of achieving a graduated progression from one size instrument to the next is not yet completely

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A   B
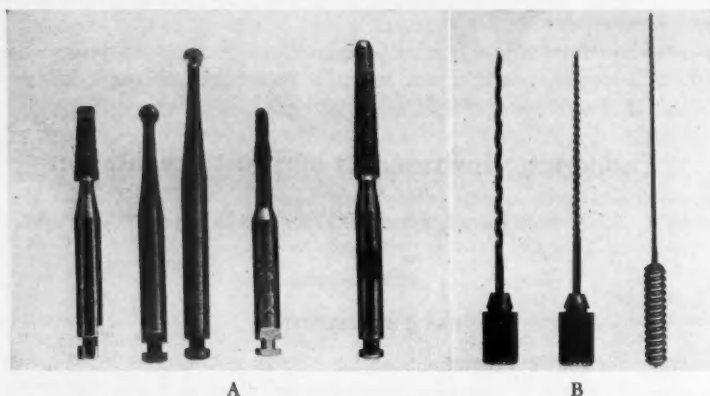
Fig. 1. *A,* Power driven instruments for coronal and intracoronal preparation. *Left to right:* Special diamond point (Stardent C1X), round burs regular length and surgical length, carbide fissure bur (Busch Widia 702U) and a special diamond point (Stardent 61-J). The latter instrument has a smooth point which will not mar the floor of the preparation.

B, Hand operated instruments for intraradicular preparation. *Left:* The reamer functions by rotation. *Center:* This file is used in a push-pull, rasping motion. *Right:* The barbed broach is primarily for pulp extirpation. Both the reamer and the file are made in long and short handles. (Courtesy, Star Dental Mfg. Co.)

solved,[2,3] reamers and files of the same number are nearly the same in size.

The Reamer. The reaming action is accomplished by rotating the instrument as it is advanced down the canal. Because they are used with a twisting motion, the smaller size reamers are easily broken in the canal if manually forced beyond the limits of the metal. Reamers are broken because they have been locked to place in the canal and then forcibly turned until the metal separates. This accident may be avoided by never rotating the bound instrument more than a quarter to a half turn. The sensation of the reamer bound in the canal may be gained by pinching one index finger between the thumb and index finger of the opposite hand and then rotating the extended finger. When an instrument "feels" this way in the canal, it should be placed under slight tension with a quarter turn rotation and then forcibly withdrawn. This is the action which cuts away the dentin wall. If the reamer is loose enough to be twirled in the canal, it cannot be broken by this action, nor, on the other hand, is it functioning. This looseness is the indication that the next sized instrument should be used.

The File. The endodontic file is used as files and rasps have always been used; that is, in a push-pull motion with the blades "set" to cut on

either the push or the pull. Files come in two different designs: either the Kerr type file with narrow "teeth" or the Hedstrom file which has the blades cut so that it resembles an inclined screw. Root canal files have the disadvantage of packing dentin filings ahead of the instrument, thus blocking the canal. Final filing must usually be followed by reaming to clear the canal.

The Broach. The third type of instrument used in root canal preparation is the barbed broach (Fig. 1B). The barbed broach has limited usefulness and is primarily employed in vital pulp extirpation, to clean debris such as necrotic material or food particles from the root canal, or to remove paper points from the canal.

INSTRUMENTARIUM

Instrument Storage. An advancement which has contributed greatly to the standardization and simplification of procedures in endodontics is the use of an instrument storage case.* The endodontic storage case has a number of advantages: (1) All endodontic instruments are stored in one central location; (2) each instrument in turn is located in a standardized position in the case; (3) the case and its contents may be readily sterilized and will remain sterile for weeks at a time.

Not only are reamers, files and broaches stored in the case, but also burs, diamond stones, paper points and cotton pellets, as well as root canal pluggers and spreaders, cement slabs and spatulas. The material is removed from the case with sterile cotton pliers, thus maintaining indefinitely the sterility of the contents. As instruments are used from the case, they are set aside. The case is then reloaded and re-sterilized when one item is finally depleted from the stock. Three to six instruments of each size are contained in the case, depending upon their frequency of use.

To prevent rusting, sterilization of these stored instruments must be done with dry heat. Sterilization at 275° F. for 45 minutes in either a sterilizing oven or a household roaster has proved quite adequate.

The Towel Kit. The standard dental instruments used in root canal therapy may be stored separately and sterilized in dental patient towels. This setup has the advantages of ready availability of sterile instruments and a sterile surface from which to work.

The kits are made up, sterilized in the dry heat sterilizer at 275° F. for 45 minutes and then stored to be used only for endodontics. Two

* Ravenna Metal Co., Seattle.

or three kits will usually suffice. The material contained within the towel kit is as follows:

(1) Two short cotton rolls used to clean instruments; (2) three 2 by 2 inch gauze sponges used to catch irrigant; (3) three Dappen dishes, one to hold cotton pellets, one for paper points and one for an irrigant such as sodium hypochlorite (Zonite); (4) a 3 cc. syringe and 25 gauge needle for irrigating the canal; (5) a straight explorer used to find canal orifices in posterior teeth; (6) a pair of operating cotton pliers used to work from the tray to the mouth; (7) a double-end spoon excavator used to remove temporary fillings, cotton pellets or debris from the chamber; (8) a front-surface mouth mirror (Kerr); (9) a Woodson plugger No. 2 used to place temporary fillings; (10) a measuring gauge (Stardent) —the gauge end to measure sterile objects, the handle end to measure roentgenograms; (11) a pair of sewing scissors used to cut paper, silver or gutta percha points; (12) a pair of sterile cotton pliers; (13) a three-well sterilizer (Kerr).

The three-well sterilizer contains tincture of Zephiran (benzalkonium chloride). The sterile cotton pliers are kept in the deep well and are used only to take cultures or to remove material from the instrument case, thereby insuring the continuous sterility of the case contents.

In the center well are stored gutta percha cones and small pieces (3 by 3 mm.) of elastic bands to be used as instrument "stops."

The towel kit is unrolled on the bracket tray or a Mayo stand. The instruments are always placed in the same position, which makes for standardization of technique. The sterile towel serves as a surface upon which to wipe instruments clean and also to clear the surface of the mirror. Even though the towel eventually becomes contaminated with the bacteria from an infected canal, this is no problem, for these bacteria are under the control of body defenses. It is the introduction of outside pathogens which must be avoided, and these pathogens are virtually eliminated with the sterile towel kit.

Rubber Dam Technique. The only way to guarantee sterility of the operating area is by using the rubber dam. For endodontics, only one tooth is usually allowed to erupt through the dam, and the clamp can be effectively placed on this tooth.

Clamp Selection. For molar teeth, a No. 18 (SSW) universal molar clamp will usually suffice; occasionally a No. 4 (Ash) clamp is needed. Most bicuspids and cuspids may be clamped with a No. 22 or No. 27 (SSW) clamp; a No. 2 (Ivory) sometimes is needed. The maxillary anterior teeth generally may be clamped with the No. 27 or No. 2 clamp; on occasion a No. 9 (Ash) or No. 00 (Ivory) clamp is used on small lateral incisors. Clamps No. 9 or No. 00 are effective on mandibular incisors.

A plastic rubber dam frame (Union Broach) made expressly for endodontics has been very helpful.

ENDODONTIC CAVITY PREPARATION

Pulp Anatomy and Coronal Cavity Preparation. Endodontic cavity preparation is divided into two phases: (*a*) coronal preparation and (*b*) radicular preparation. The ultimate aim in endodontic therapy is the instrumentation and obturation of the root canal space, but to make this possible, the coronal preparation must be correct in size, shape and inclination.

The relationship between endodontic cavity preparation and pulpal anatomy is so flexible that both subjects must be studied together. The operator must always have in mind a three dimensional image
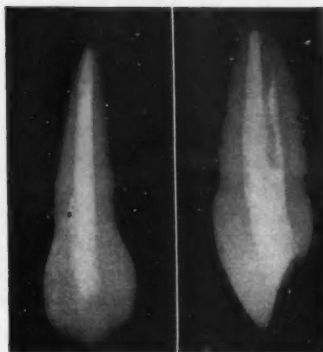
Fig. 2. Failure of roentgenogram to reveal the third dimension. *Left:* Conventional labiolingual view of a laboratory exercise apparently demonstrates a well filled canal. *Right:* Mesiodistal view reveals the breadth and how poorly obturated is this ovoid canal.

of the inside of the tooth, from pulpal horn to apical foramen. The roentgenogram provides a two dimensional blueprint of pulpal anatomy and deserves preoperative attention, but it fails to reveal the entire story because it lacks the third dimension (Fig. 2).

A modification of G. V. Black's principles applied to *endodontic* cavity preparation is developed by deleting Black's "retention form" and "finish the enamel wall" and adding to the principles the final stage of endodontic preparation, *canal instrumentation,* thus: (1) outline form; (2) convenience form; (3) removal of remaining carious dentin (and defective restorations); (4) toilet of the cavity; (5) canal instrumentation.

Principle 1—Outline Form

The outline form of the endodontic cavity must be correctly shaped and positioned to establish complete access for instrumentation from

pulpal horn to apical foramen. Since external outline form is related to the internal pulpal anatomy, endodontic preparations must be done from the inside of the tooth to the outside. External outline form is established during preparation by mechanically projecting the internal anatomy of the pulp out onto the external surface. This may only be accomplished by drilling into the open space of the pulp chamber and then working with the bur from inside the tooth to the outside, cutting away the dentin of the pulpal roof and walls which overhangs the floor of the chamber. The tendency to establish outline form in the conventional operative manner and then drill into the chamber must be resisted.

To achieve the optimal preparation, three factors of internal anatomy must be considered: (a) the size of the pulp chamber, (b) the shape of the pulp chamber, and (c) the direction or curvature of the individual root canals.

The size of the pulp chamber materially affects the outline form of endodontic access cavities. In young persons these cavities must be more extensive than in older persons where the pulp has receded and the pulp chamber is smaller in all dimensions. In the anterior teeth of youngsters, the large root canals require large filling materials for obturation—materials which in turn will not pass through a small orifice in the crown.

The shape of the pulp chamber should be accurately reflected in the finished outline form; e.g., the triangular shape of the floor of the pulp chamber in a molar tooth is extended with the walls of the cavity out onto the occlusal surface, hence the final molar cavity outline form is triangular in shape. As another example, the coronal pulp of a maxillary premolar is flat mesiodistally but elongated buccolingually, so the outline form is an elongated oval which extends buccolingually rather than mesiodistally as the Black's operative cavity preparation.

The curvature and direction of the root canal is the third factor regulating outline form. For efficient instrumentation without interference, the cavity walls often must be extended to allow an unstrained instrument approach to the apical foramen, and this extension affects the outline form.

Principle 2—Convenience Form

In endodontic therapy, convenience form makes more convenient and more accurate the instrumentation as well as the filling of the root canal. Convenience form is necessary for unobstructed access to the canal orifice, for direct access to the apical foramen, and for complete control over the enlarging instrument.

Unobstructed Access to the Canal Orifice. In endodontic cavity preparation of all teeth, enough tooth structure must be removed to allow instruments to be easily placed into the orifice of each canal without interference from overhanging walls. The orifices must be visible and easily reached with instrument points (Fig. 3A), otherwise the results are endangered and treatment time is increased.

Direct Access to the Apical Foramen. To provide direct access to the apical foramen, enough tooth structure must be removed to allow the endodontic instruments freedom within the coronal cavity so that

A   B

Fig. 3. *A,* Unobstructed access to canal orifice. The mesial wall has been sloped toward the mesial. A curved instrument, with the curved tip pointed toward the mesial, travels down the mesial wall until the tip engages the orifice. The instrument is then given a one-half turn so that the instrument curves in the same direction as the canal. The distal canal orifice is easily entered from the mesial. Note that the distal cavity wall is not vertical but slopes mesially.

B, Severely curved canal successfully instrumented and obturated. *Left:* Note the bayonet curve. *Right:* "Trial cone" view. Note how far to the mesial the access cavity was prepared to give more direct access to the curved canal.

they may extend down the canal in a direct line. This is especially true when the canal is severely curved or leaves the chamber at an obtuse angle (Fig. 3B). Gross extension is also necessary in many molars—the buccal wall in maxillary molars and the mesial wall in mandibular molars; occasionally, even decuspation is undertaken.

Complete Control of the Enlarging Instrument. The clinician must maintain complete control over the direction of the instrument. For this reason, tooth structure is removed around the canal orifice so that the instrument stands free in this area of the canal and is controlled only by (1) the clinician's fingers on the handle of the instrument, and (2) the walls of the canal at the tip of the instrument. Nothing is to intervene between these two points.

Violation of any of these factors related to convenience form will ultimately lead to failure by either root perforation, "shelf" formation within the canal, instrument breakage, or incorrect shape of canal instrumentation, i.e., slit-shaped preparation.

Individual Coronal Preparations

The principles of endodontic coronal preparations are discussed below for maxillary central incisors, maxillary first premolars, maxillary first molars, and mandibular first molars. The information may then be applied to anatomically similar teeth.

The Maxillary Central Incisor. The principles of preparation for the maxillary central incisor will apply to all of the anterior teeth. Entrance into the pulp chamber of anterior teeth is *always* through the lingual surface. Proximal penetration is not direct line access to the canal and perforation may result.

Initial entrance into an anterior tooth is made exactly in the center of the lingual surface. If the lingual surface is mentally divided into squares with the familiar "tic-tac-toe" frame, then the cavity is begun in the center square. The usual error is to begin the cavity too far gingivally.

Enamel penetration is best accomplished with a tapered diamond point (Starlite C1X) or carbide bur (Busch Widia, 702U) in a contra-angle. Initially the instrument is operated at right angles to the long axis of the tooth until the enamel is perforated (Fig. 4A). Then, without stopping, the position of the contra-angle is changed so that the stone is operated parallel to the long axis of the tooth. The cavity is thereby extended incisally to satisfy the need for convenience in later entering the pulp chamber (Fig. 4B). This extension is developed with slight "fanning" of the preparation mesially and distally. The bevel toward the incisal should not be so long that the surface cannot readily be restored. Only the enamel should be prepared. At no time should the tapered stone be "forced." Forcing a tapered instrument may "check" or crack the enamel.

The external preparation is relatively complete with this extension and the next step of preparation is the entrance into the pulp chamber through this cavity. It is necessary, in entering the pulp chamber, to be able to operate the shaft of the bur in the direction of the long axis of the tooth. Removal of lingual enamel toward the incisal (lower arrows, Fig. 4C) carries the preparation further labially and allows the shaft of the bur (No. 4) to enter directly into the long axis of the pulp chamber (Fig. 4D). Gingival extension (upper arrows, Fig. 4C) carries the cavity away from the central axis. Failure to extend the
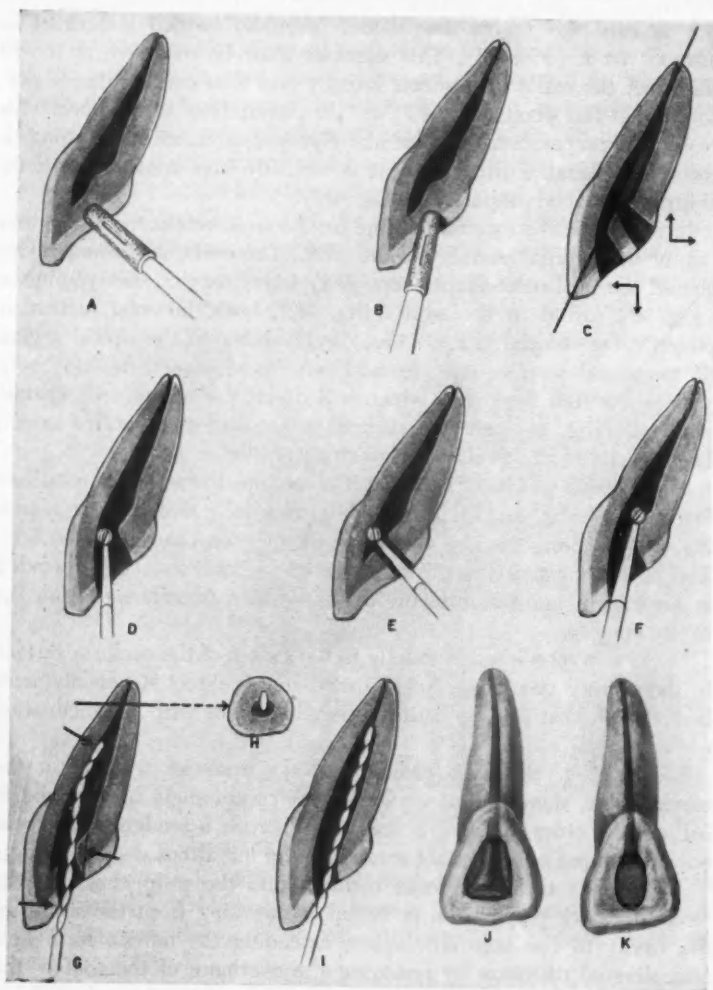
Fig. 4. (See text for explanation.)

initial preparation incisally will cause the opening bur to be directed labially, and ditching of the labial dentin or even labial perforation may result (Fig. 4E). In addition, instrumentation of the canal through this limited cavity would be most ineffective.

As soon as the bur "drops" into the pulp chamber, the internal preparation is begun, *working from the inside out*, removing the linguo-proximal walls of the chamber. The No. 4 surgical length

bur is used to remove the lingual "shoulder" which is present in anterior teeth (Fig. 4F). This shoulder must be removed, or it will misdirect the canal instruments labially and thus cause either a perforation at this point (Fig. 4G) or "slit preparation" at the apex. This restrained preparation does not allow proper mechanical cleansing of the apical canal. Furthermore, it is virtually impossible to obturate this pestle-shaped preparation (Fig. 4H).

In summary, the external outline form should reflect the shape and size of the internal anatomy of the tooth. The eventual coronal cavity should be a funnel-shaped opening, triangular in the youngster (Fig. 4J), ovoid in the adult (Fig. 4K), both, however, extended incisally for convenience, so that direct access to the apical region of the canal is adequate. In addition, there should be no tooth structure within the cavity which will divert the instruments against one wall (Fig. 4I), and the external orifice and canal orifice should be adequate in size to allow optimum obturation.

The Maxillary First Premolar. The outline form in the maxillary first premolar is ovoid in shape buccolingually and narrow mesio-distally, reflecting the size and shape of the pulp chamber (Fig. 5A). The limits of extension will be governed by the convenience needed to instrument and obturate the canals without interference from the cavity margins.

Initial entrance is made exactly in the center of the occlusal surface in the central groove. A No. 4 round bur is directed apically, care being taken that the bur is in correct alignment with the inclination of the crown.

The floor of the pulp chamber in the posterior teeth is at the cervical line, therefore, a *regular length* contra-angle bur will be in full depth before the floor is reached. There is a tendency to expose only the pupal horns by not extending the initial cut deeply enough.

As soon as the bur breaks through into the pulp chamber, the internal cavity preparation is begun by working from the inside of the cavity to the external surface, extending the outline form in a buccolingual direction by removing the overhang of the roof of the chamber. No dentin should be removed from the lateral walls of the cavity.

When this ovoid cavity extension has progressed to about double the size of the entering bur, then the floor of the cavity should be investigated with an explorer for the canal orifices, to determine the direction of further extension. The explorer should be followed by a No. 1 style B reamer placed well down each canal. If the reamer is pressed tightly against the buccal or lingual walls of the cavity, the extension should be made in the direction which will free the action

of the instrument (Fig. 5B). Frequently, the entire roof of the pulp chamber need not be removed vertically in the maxillary first premolar if the pulpal horn area can be cleansed and if the instruments can reach the apex unimpeded by cavity margins. This is especially true if the tooth has two widespread roots (Fig. 5C). If the roots are parallel, the outline form must be extended further buccolingually to accommodate instrumentation (Fig. 5D).

The Maxillary First Molar. The outline form on the occlusal surface of the maxillary molars is roughly triangular in shape with the apex of the triangle toward the lingual, reflecting the "molar triangle" of
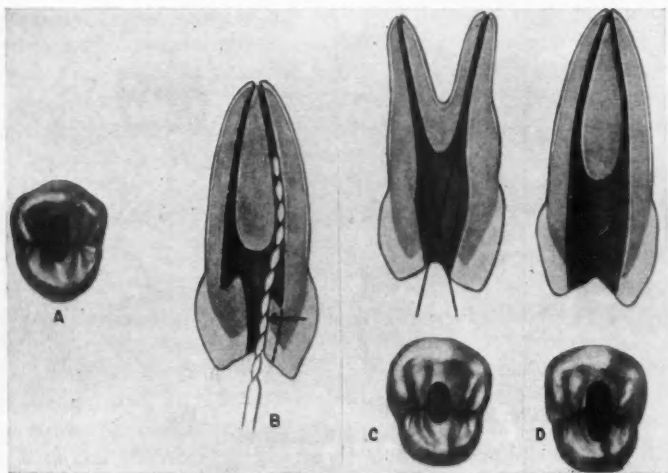
Fig. 5. (See text for explanation.)

the pulp chamber (Fig. 6A). It should again be emphasized that this form is established by extending the size and shape of the chamber onto the occlusal surface, there to be modified for convenience.

Initial entrance is gained through the mesial central pit with a No. 4 or No. 6 round bur inclined slightly lingually. This will direct the bur into the large orifice of the lingual canal (Fig. 6B). Guided by tactile sensation, the roof of the pulp chamber is removed with the round bur cutting away the overhanging dentin toward the buccal (Fig. 6B).

Before too much tooth structure is removed, an explorer should be used to find the orifices to the canals. This knowledge will assist in developing extension. The usual tendency in molar preparation is to over-

cut the cavity, which is easily done with a No. 8 or No. 11 bur. The oblique ridge need not be invaded nor is it necessary to extend the outline form lingually as far as the lingual canal orifice. The lingual canal usually leaves the floor of the chamber at about a 60 degree angle. Because instrumentation of this canal is from the buccal, it is not necessary to develop vertical access into the lingual canal (Fig. 6C).

The orifices of the two buccal canals lie well to the buccal and join the chamber at a right angle to the floor. Convenience form calls for flaring the preparation buccally. This extension is made with a
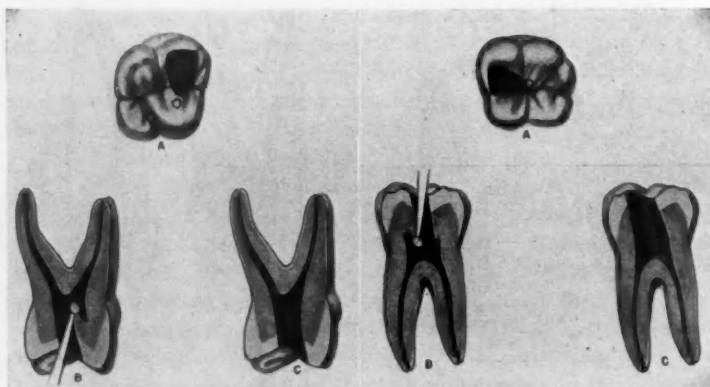
Fig. 6.   Fig. 7.
(See text for explanations.)

tapered fissure bur (702U) or diamond stone (61-J) (Fig. 6C). Sloping the buccal wall assures rapid access to the buccal orifices, control of the endodontic instruments and prevention of "shelf" formation in the canal.

It is important to note that the entire preparation may be kept in the mesial half of the tooth and that the distal buccal orifice is at the buccal midline and not under the distobuccal cusp (Fig. 6A).

The buccal canal orifices must be completely within the floor of the preparation with no dentin of the lateral walls surrounding the orifice. A No. 2 bur is used to complete this portion of the preparation.

The Mandibular First Molar. The outline form on the occlusal surface of mandibular molars is triangular in shape with the apex of the triangle toward the distal, again reflecting the "molar triangle" anatomy. The preparation should be completely within the mesial half of the tooth (Fig. 7A).

Initial entrance is made just mesial to the central pit with a No. 4 or No. 6 carbide bur, inclined slightly distally. This will direct the bur into the large orifice of the distal canal. The roof of the pulp chamber is then removed with the round bur cutting away the overhanging dentin toward the mesial (Fig. 7B).

Early in preparation an explorer should be used to find the orifices of the three canals, then extension toward the mesial may be more exacting. Outline form need not be extended distally past the midline, for the distal canal leaves the chamber at an angle and instrumentation is from the mesial.

On the other hand, the orifices of the two mesial canals lie well toward the mesial and join the chamber at a right angle to the floor. Convenience form therefore calls for flaring the preparation to the mesial. This extension is made with a tapered fissure bur (702U) or diamond stone (61-J) (Fig. 7C). Sloping the mesial wall assures unimpeded progress with the endodontic instruments and prevents perforation or "shelf" formation within the canal.

The mesial canal orifices must be completely within the floor of the preparation with no dentin of the lateral walls surrounding the orifice. A No. 2 bur is used to complete this portion of the preparation.

Principle 3—Removal of Remaining Caries and Defective Restorations

After outline form is established and prior to canal preparation, all remaining caries and defective restorations are to be removed from the crown. If caries, contaminated with bacteria, is allowed to remain in the chamber, it will be virtually impossible to sterilize the pulp canal. Furthermore, a failing restoration, leaking at the margins, will allow continuous bacterial contamination of the pulpal space.

These badly broken down teeth may be restored with cement or temporarily rebuilt with amalgam or a copper or orthodontic band.

Principle 4—Toilet of the Cavity

Toilet of the cavity is an essential step in endodontic preparation just as in operative dentistry. Debris left remaining in the crown will eventually stain the tooth or may also be highly contaminated with bacteria. Furthermore, there is the constant possibility of crumbs of cement or amalgam dropping into and occluding the root canal, particularly in the mandibular teeth.

In addition to the air blast and round burs, spoon excavators or periodontal curets are used for maintaining cavity toilet. Final debris

is always irrigated from the canal and chamber by sodium hypochlorite lavage.

Establishing the Length of Tooth

Before any canal enlargement is undertaken, an accurate length of tooth should be established. This may be done quite simply with a reamer, a ruler and a roentgenogram, as shown and described in Figure 8.
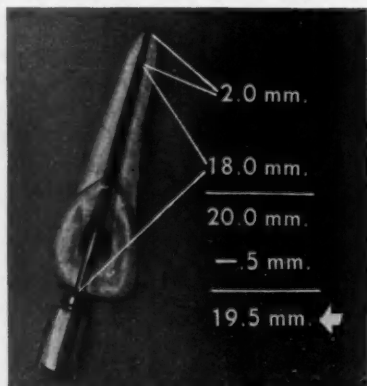
Fig. 8. Accurate determination of tooth length. First measure tooth in original roentgenogram and subtract 2 or 3 mm. from that tentative length to compensate for distortion and avoid forcing of instrument through apex. Place a style B reamer in the canal to that depth (in diagram, 18 mm.) and take another roentgenogram, which will reveal distance of point of instrument from foramen (in diagram, 2 mm.). Sum of these two figures (here 20 mm.) is total tooth length. However, to compensate again for distortion a safety factor of 0.5 mm. is subtracted from over-all length, leaving the "working length" (in diagram, 19.5 mm.). Good evidence indicates that this is the ideal point at which to terminate endodontic procedures.[1,4]

Intraradicular Preparation: Pulpectomy

If vital pulp extirpation is necessary, the barbed broach is slowly inserted into the body of the pulp tissue as far down the canal as possible, is slowly rotated one full turn and then is slowly removed. Broaches are easily broken, therefore, one must use care that the broach is not lodged in the dentin walls. Moreover, the operation must be done slowly so that the broach entwines the pulp tissue and does not tear it.

The largest possible broach should be used in each case of pulpectomy and may be selected for diameter and length by comparing the instrument as a silhouette against the initial roentgenogram.

If the canal is curved the broach should not be twisted more than one quarter turn.

Extremely large pulps cannot be extirpated with a single broach. In these cases, two or three broaches must be intertwined in the pulp to complete the pulpectomy.

It is not generally necessary to use a broach for pulpectomy in canals of very small lumen. In these cases, canal instrumentation with the reamers and files concomitantly removes the pulp.

Principle 5—Canal Instrumentation

Root canal preparation has two ultimate goals, the mechanical cleansing of the root canal space and the preparation of that space for total obturation, i.e., preparation of a round, tapered canal space with a minimal apical opening.[5] Fortunately both goals are accomplished by the same procedure of instrumentation.

Mechanical Cleansing of the Canal. One can be certain that bacteria are eliminated only through the use of the bacterial culture; pure mechanical instrumentation will immediately sterilize relatively few infected canals. This is why instrumentation is followed by intracanal sterilization with drugs.

On the other hand, when the reamers are removed from the canal after each use, and clean, dry, white dentin chips are found clinging to the apical 3 or 4 mm. of the instrument, the operator realizes that he is rapidly approaching optimal dentin removal. The tip of the reamer is inspected to determine progress. The instrument must be cleaned in a cotton roll each time it is removed from the tooth.

When the files are used, the dry, clean, powdered dentin filings fall to the rubber dam or gather in the chamber. When discolored moist dentin may no longer be removed and when the canal walls "feel" glassy smooth to the file, the canal filing may be considered complete. The canal and chamber should then be irrigated with sodium hypochlorite and dried with paper points.

Canal Preparation for Obturation. Anatomically, most root canals tend to become round in diameter in the apical 3 or 4 mm. Because the filling materials used in endodontics are round, better obturation can be obtained with these materials if a round "seat" or "home" is prepared for their reception. This round, tapered apical "seat" is best developed with a reamer, because the reamer will drill out the uneven dentin walls to an even bore the approximate size of the preformed filling material (Fig. 9).

Owing to their anatomic shape, a few canals are generally enlarged entirely by reaming. These canals are (*a*) the small canals in the

molars (buccal canals in the maxillary molars and mesial canals in the mandibular molars) and (*b*) the canals in the maxillary first bicuspid. All other root canals usually require some filing, because the shape of most root canals is a greater flare than the taper of the reamer used in apical preparation. The files must, therefore, be used to enlarge the ovoid portion of the canals where the reamers are ineffective. This filing procedure is done in the shape of the Maltese cross, that is, the
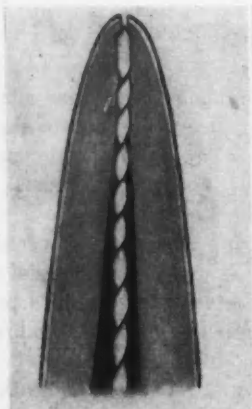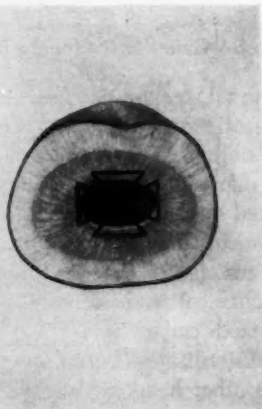
Fig. 9.   Fig. 10.

Fig. 9. Reaming out the apical 3 or 4 mm. of the canal to develop a round, tapered "seat" in preparation to receive the round, tapered filling materials. The remainder of the canal must be filed. Note the preparation terminating 0.5 mm. short of the foramen.

Fig. 10. "Maltese cross" filing superimposed on the cross section of an ovoid canal. The file must be forced against the walls in all directions, in the pattern of a Maltese cross.

file is forced against the walls of the canal in all four directions (Fig. 10). If the file is curved gradually, it may be more easily manipulated in this operation.

Filling materials are inaccurate in size and shape when compared to endodontic instruments.[3] One manufacturer is attempting to standardize filling material size and instrument size although the ultimate goal has not been achieved. Shape of the filling materials has at least been changed to more nearly approximate the taper of the preparing instrument.

There recently has been developed a gauge which will allow accurate comparisons between the last instrument to be used in preparing the canal and the initial point to be used in obturating the canal.[6]

Rules Governing the Use of Reamers and Files

The following rules should be followed during canal enlargement:

1. Always curve the point of the instrument (Fig. 11). This gives the operator more control over the instrument for it allows him to scribe a circle with the point, not have it only pivot on center.

2. Always use a curved instrument in a curved canal. A straight instrument will immediately cut a shelf in a curved canal (Fig. 12).
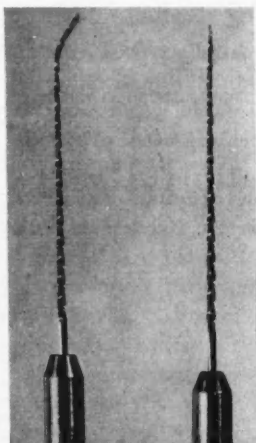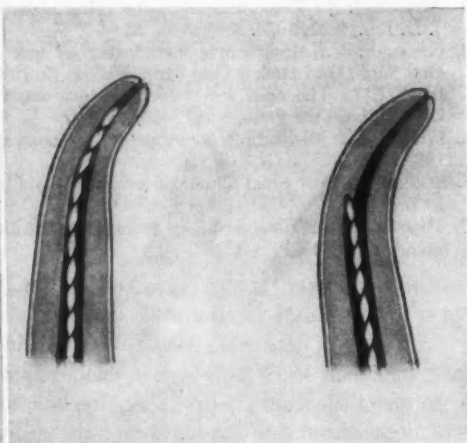
Fig. 11.   Fig. 12.

Fig. 11. *Left:* Curved tip of the exploring reamer. The operator may truly explore the canal walls with this curved tip by turning and probing with the instrument. *Right:* The perfectly straight instrument. The tip rotates on the axis of the instrument and the operator cannot probe or explore an uneven surface with this tip.

Fig. 12. The importance of using a curved instrument in a curved canal. *Left:* Note how the flexible curved instrument follows the curve of the canal. *Right:* The *straight* instrument catches in the uneven canal wall and begins shelf formation which may ultimately lead to perforation. (After Pucci.)

3. Always restore the curve to an instrument after each use.

4. Constantly clean the instruments as they are being used.

5. Always use instruments in sequence of size. Attempting to "jump" sizes will often lead to "shelf" formation.

6. Always use the instruments to their full measured length before advancing to the next size instrument.

7. Always enlarge very fine canals with reamers *and* files, advancing slowly from one size to the next.

8. Never rotate an instrument more than a quarter turn if it binds in the canal.

9. Always limit canal instrumentation to a point 0.5 mm. short of the apical foramen.

10. Always irrigate the debris from the canal with sodium hypochlorite or like solution.

Anatomic drawings in this article after Zeisz and Nuckolls.

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Rational Root Canal Medication

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In order to retain a tooth in a healthy condition, if the pulp has been infected, we must first remove the organic pulp tissue from which microorganisms may gain sustenance. If possible, we should at the same time remove the microorganisms therein contained.

The objective of this procedure is to reduce the toxic by-products of tissue breakdown, which, after isolation and purification by Menkin, have been called necrosin.[8] Necrosin can produce breakdown of both soft and hard tissues. If a periapical lesion is present in a tooth that has no visible caries, when a culture is taken we frequently find that the pulp is free of microorganisms. This area of rarefaction is a by-product of the tissue breakdown substances (necrosin).

This emphasizes the importance of removing all of the tissue from within the root canal, to prevent periapical pathosis. Elimination of these substances from within the root canal and its immediate vicinity brings the tissues within physiologic equilibrium and repair or healing may be expected of the supporting tissues.

Thus, rational medication begins with thorough chemo-mechanical preparation of the canal. Auerbach has demonstrated that by careful mechanical cleansing and irrigation of the canal alone, without use of further medication, 78 per cent of the infected teeth thus treated yielded growth-free cultures.[1] The author, in a somewhat modified study, found that 76 per cent of the infected teeth which were treated by chemo-mechanical preparation alone were free of microbial growth. This percentage was calculated after two consecutive negative cultures were obtained.[14]

These findings indicate that medication, although a valuable adjunct, is not the most important phase of root canal therapy. However, medication properly selected and judiciously used can be a great aid in helping achieve the objectives of root canal therapy. Properly selected and carefully applied medication can eliminate the microorgan-

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isms which remain in the root canal after the chemo-mechanical preparation has been completed. It could also help eliminate organic matter and act as a detoxifying agent in the preparation of the root canal.

Buchbinder and Bartels have demonstrated the evidence of natural antibiotic substances that are present in the tissue exudates found in infected root canals.[2,3] They found that these fluids probably contain a lysozyme, which they demonstrated was present when infection was
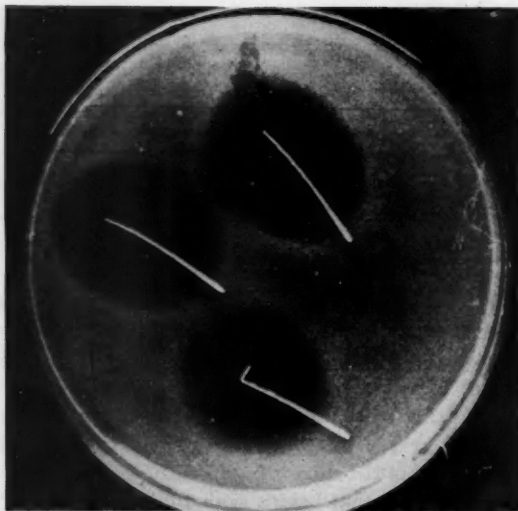
Fig. 1. The effect of natural antimicrobial substances (possibly lysozyme) isolated from infected root canals. The pour plates were prepared with 1 per cent infusion agar seeded with *Micrococcus lysodeikticus*. (Courtesy of Dr. Maurice Buchbinder.)

evident. Figure 1 shows the zones of inhibition produced by the exudate in seeded Petri plates.

Other substances in the blood, including the various phagocytic cells, aid in destruction and digestion of the microorganisms. Also, the role of the lymphocytes in the production of detoxifying agents is well known. Therefore, it is important to use only those drugs that will not inhibit the natural defense mechanisms of the body for healing and repair.

MEDICATION

The one ideal medicament for treating infections, not only in the root canal but in the rest of the body as well, is at present not known,
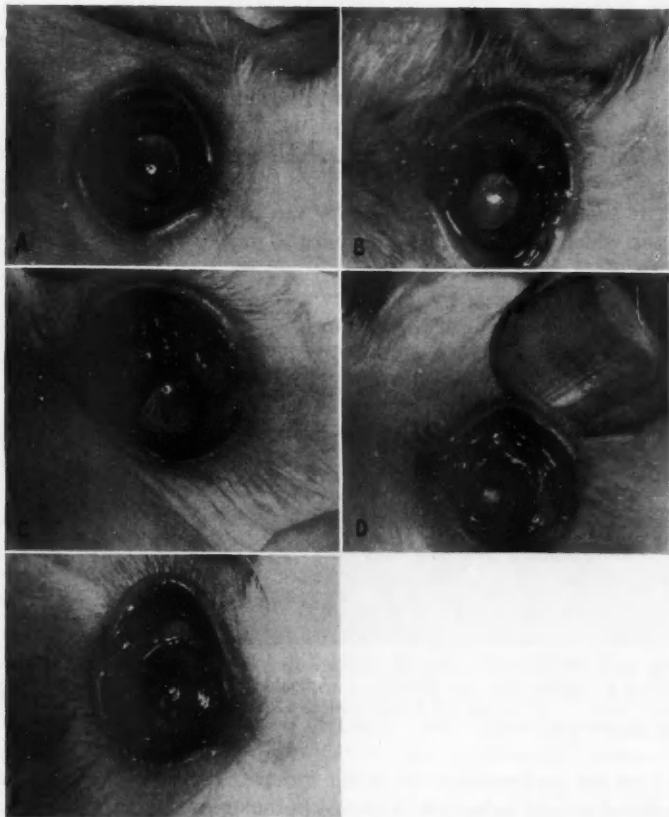
Fig. 2. *A*, Normal eye of rabbit. *B*, Rabbit eye exposed to eugenol shows irritation, swelling, change in cornea. *C*, Rabbit eye 3 to 5 minutes after exposure to *p*-monochlorophenol; note marked edema and change in cornea. *D*, Rabbit eye 3 to 5 minutes after exposure to PBSC polyantibiotic; note edema and irritation. *E*, Rabbit eye 3 to 5 minutes after exposure to antihistamine-antibiotic preparation; note inflammation and changes in cornea.

although there is a vast number of new medications constantly reported upon in the hope of achieving this goal. Let us then list the characteristics of an ideal root canal medicament.

1. First of all, it should be effective in eliminating or destroying those microorganisms which we normally find within the root canal.

2. It should destroy, or neutralize, or eliminate any toxic products which may be present within the root canal.

3. It should be non-irritating to healthy tissue and not produce toxic or allergic reactions.

4. It should not stain or discolor the tooth structure.

5. It should have good penetrating qualities, in order to be effective deep within the dental tubules.

6. It should remain stable at room temperature for long periods of time.

7. It should not inactivate the natural defense mechanisms of the body.

8. It should not be inactivated by the blood, or by serum protein or pus which may be present within the root canal or the periapical tissues.

9. It should be readily available.

10. It should be easy to apply.
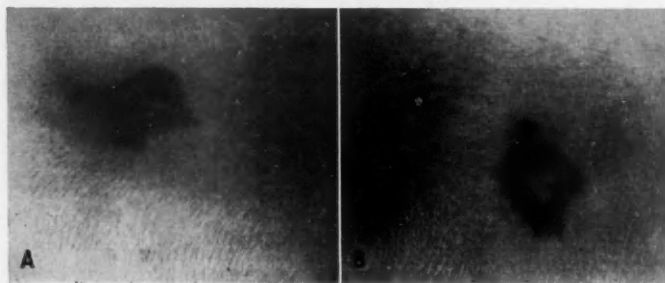
Fig. 3. A, Rabbit skin one day after eugenol injection; note inflammatory reaction. B, Rabbit skin one day after p-monochlorophenol injection; note inflammatory reaction. (The author is indebted to Dr. Herbert Schilder of Boston for these photographs.)

There are at present three main groups of substances commonly employed in root canal medication: the halogens, the phenol derivatives, and the antibiotics. We may possibly consider as a fourth category, a combination of these substances.

Although various compounds used for many years have been considered non-irritating, most of the substances which are commonly used today will produce some degree of inflammatory action.[10] (See Figs. 2 and 3.)

We must realize, however, that in root canal medication we are dealing primarily with topical medication, and the compounds used are intended to remain in place within the root canal, against the hard surfaces. Medication leaking beyond the apex will come in contact primarily with connective tissue substances, and the irritating properties of the medication would not be so severe as in the delicate epithelial tissues of an eye. However, the fact remains that the compounds which are now commonly being used do have some

inflammatory potential. It is, therefore, important that the mechanical preparation of the canal be done judiciously and that the tissues be treated gently at all times, in order not to aggravate the situation further, although for the most part the inflammatory reactions are mild and healing will usually follow.

Let us now consider the various groups of compounds which were indicated above.

Halogens

The halogen elements are bromine, chlorine, fluorine and iodine. For disinfectant purposes in the root canal, the most commonly employed are the chlorine-liberating substances and iodine compounds.

Chlorine. Because of the instability of the chlorine compounds, they would not be suitable to seal in a root canal, but are used primarily in the chemo-mechanical preparation of the canal. Chlorine has the advantage of being a bleaching agent, and its alkaline solutions have a solvent action upon necrotic pulp tissue.[4] In the double strength sodium hypochlorite or the standard formula, Labarraque's solution, this is particularly evident.

A commercial chlorine preparation which is a bit more stable and readily obtainable is Zonite; it can be used full strength and is equivalent to the double strength chlorinated soda solution. The reaction of hydrogen peroxide with sodium hypochlorite in the root canal yields sodium chloride, water, and oxygen, which are used in flushing the canal and detoxifying and digesting disintegrated protein matter.

Camphorated Parachlorophenol. Camphor and parachlorophenol are both solids, but when they are mixed in correct proportions, they liquefy. The official preparation contains 35 parts of parachlorophenol and 65 of camphor, both by weight. According to Prinz,[9] a mixture of this type was recommended by Walkhoff in 1891 for use in root canal therapy. More recently, Sommer, Ostrander and Crowley have suggested that it be combined with soluble penicillin for treatment of infected root canals.[12] Their results have been excellent. The parachlorophenol is relatively non-irritating, particularly when one compares it with formocresol, which will be discussed shortly.

Iodine Solutions. Iodine is another halogen which is used as a root canal medicament, primarily in a zinc iodide solution. It has been used in conjunction with electrolytic sterilization. This technique of medicating the root canal, although effective, has certain disadvantages. The iodine tends to discolor the tooth structure, it is sometimes highly irritating to the periapical tissues, and the treatment is quite time consuming.

Phenols

Liquefied phenol has for some time been used to destroy micro-organisms within the root canal. It is a powerful caustic, however, and as such has certain limitations, though it might be useful in coagulating the protein and remaining pulp fibers after the extirpation of a vital pulp. However, because of its inability to penetrate deeply within the dentin structure it is not recommended as a routine antiseptic.

Formocresol. This mixture is occasionally used to disinfect the root canal. However, it is a highly irritating substance, and may irritate the periapical tissues and produce a great amount of exudate as well as persistent pain. It is, therefore, not recommended for root canal treatment.

Cresatin. Cresatin, or metacresyl acetate, has a low surface tension and penetrates deeply. It is non-irritating, according to Schilder,[10] and has excellent antiseptic qualities. It is also used because of its fungicidal properties, when root canals are infected with monilia.

Antibiotics

With the introduction of the antibiotics, the practice of endodontics became more popular, since root canals could now be quickly and safely freed of microorganisms.

The antibiotics should be reserved for use in putrescent pulps, and for chronic or acute abscesses. In instances where vital pulp extirpations have been performed, their use is not justified. Other forms of medication, as have been discussed, are adequate to destroy the relatively small numbers of microorganisms which are present in these instances.

The indiscriminate use of antibiotics constitutes their primary danger. There is the possibility of creating resistant forms of microorganisms, and when penicillin, particularly, might be needed for a more serious type of systemic involvement, it would have consequently been rendered ineffectual.

We must also consider the possible sensitivity of a patient to the antibiotic, and future allergic manifestations as a result of its use. Figure 4 shows one such dermatologic response to penicillin, applied only in the root canal. In cases of known sensitivity to an antibiotic, its use should be avoided and some other form of medication should be employed. We should also exercise caution when a patient has any allergic history at all.

Penicillin, used alone, is now probably obsolete as a substance to be sealed into the root canal, or even as an irrigating medium, since it is
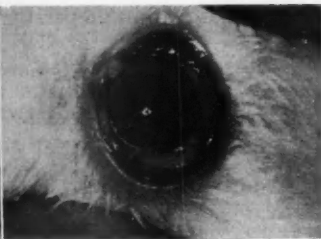
Fig. 4. Allergic reaction to penicillin in root canal medication. Note scaly dermatitis.

Fig. 5. Rabbit eye; no apparent irritation 3 to 4 hours after instillation of lidocaine ointment (5 per cent). (Courtesy of Dr. Herbert Schilder of Boston.)

known that there are many different types of organisms found in the root canal. Frequently gram negative colon organisms are isolated from the canal. These produce a penicillinase which will inactivate penicillin.

Also in many instances, we may have a number of different types of microorganisms within one root canal. One drug, alone, would not be effective in these cases. Therefore, various medicaments, in combination with penicillin, or containing substances other than penicillin, have been introduced.

One of the first of this group of polyantibiotic compounds to be used successfully is Grossman's PBSC.[5] This represents

Potassium penicillin G	1,000,000	units
Bacitracin	10,000	units
Dihydrostreptomycin	1.0	Gm.
Sodium caprylate	1.0	Gm.
Silicone liquid DC 200	q.s.	

This preparation is now commercially available. It is a thick paste that can be introduced into the pulp chamber or canal by means of a syringe. It has been homogenized and is stable, even at room temperature.

Although this material has proved quite successful in the elimination of microorganisms from within the root canal, it cannot be prepared in the dentist's office, and the bacitracin does produce some degree of periapical irritation.

Seltzer and Bender[11] modified the formula by substituting chloramphenicol for the bacitracin, and by using procaine penicillin. The paste which they prepared uses the liquid that is normally supplied in the disposable cartridge of procaine penicillin as its vehicle.

The formula for this preparation is as follows:

Procaine penicillin G (aqueous)	300,000 units
Chloramphenicol	250 mg.
Streptomycin calcium chloride complex	250 mg.
Sodium caprylate	250 mg.

This paste can be prepared in the dental office, and has proved quite successful clinically.

A further modification was introduced by the author in the form of an antihistamine-antibiotic mixture.[15] The formula for this D.C.P. cream in its improved form is as follows:

Benzathine penicillin G	300,000 units
Chloramphenicol (crystalline)	125 mg.
Chlorcyclizine (antihistamine)	100 mg.
Lidocaine ointment 5%	0.5 cc.

Clinically this cream has been highly successful, both in eliminating the microorganisms within the canal and in reducing the amount of postoperative discomfort.

It may be readily prepared in the dentist's office by placing four chlorcyclizine tablets (50 mg. each) in a sterile mortar, as used for mixing amalgam, and triturating to a fine powder. Then the contents of a 250 mg. capsule of chloramphenicol are added and triturated further. Next the benzathine penicillin, in a 1 cc. disposable cartridge containing 600,000 units, is added and a paste-like mass produced. The lidocaine ointment is finally added and the mixing continued until a smooth creamy consistency is obtained.

The cream is stored in a 5 cc. Luer-Lok syringe or in anesthetic cartridges, where it is stable for about one year at room temperature. It has the additional advantages of containing lidocaine ointment and an antihistamine, both of which may reduce periapical sensitivity. In addition, the chlorcyclizine may inhibit the growth of fungi and yeast-like organisms within the canal, as well as help prevent possible allergic reactions to the antibiotics.

The polyantibiotic pastes have been criticized by some because they tend to invalidate the culture technique.[3] It has been stated that trace amounts of the antibiotics which are carried into the culture tube are not inactivated, and so produce false negative cultures.

The clinician, however, will be impressed by the fact that only trace quantities of an antibiotic are sufficient to inhibit bacterial growth in a culture tube. Might we not then logically expect concentrated quantities of the medication, in the root canal, to be eminently more effective in destroying microorganisms?

Grossman, Parris and Cobe[7] have demonstrated that when a poly-antibiotic paste is used in the root canal, if the canal has been thoroughly dried with four absorbent root canal points, the amount of material which is carried over into the culture medium, which might inhibit bacterial growth, is so minute that the possible number of false culture readings would become statistically insignificant.

As mentioned earlier, Sommer, Ostrander, and Crowley[12] have advocated the use of a penicillin-camphorated parachlorophenol mixture in highly infected root canals. This is prepared immediately before use, by placing a 50,000 unit tablet of soluble penicillin on a sterile glass slab, adding a drop of camphorated parachlorophenol and then spatulating. This produces a smooth creamy paste which can be easily introduced into the root canal on a file or reamer. Excellent clinical results have been obtained with this mixture, and it has one advantage over the polyantibiotic mixtures, for if a penicillinase is used in the culture medium, the antibiotic does not interfere with accurate culture technique.

In cases of a known penicillin sensitivity in which the use of an antibiotic is essential, one of the broad spectrum antibiotics may be used instead. The author has successfully used chlortetracycline, tetracycline and chloramphenicol in these cases.

In Figure 5 the effect of lidocaine ointment upon the eye of a rabbit is shown. It is so well tolerated that the author has used it as a vehicle for antibiotics.

If the antibiotic is dispensed in a capsule form, the capsule is cut with a scalpel, and the contents placed on a sterile slab. A small quantity of the lidocaine ointment is then spatulated in the mixture to form a creamy mass. If the antibiotic is in a tablet form, it is carefully pulverized with a pestle in a small mortar such as is used in the preparation of amalgam. However, it is most important that there be no amalgam present on the mortar and pestle, since the heavy metal in the amalgam will tend to inactivate the antibiotics.

It can not be too strongly emphasized that no compound can be really effective unless the canal is thoroughly cleansed and prepared beforehand.

SEALING MEDICAMENTS WITHIN THE CANAL

The most widely used means of placing medicaments in the root canal is the absorbent point. After careful preparation of the canal, which includes thorough mechanical instrumentation and irrigation, the canal is dried, with either electric drying points or sterile dry absorbent points. The medicament of choice is then sealed in the

canal, either by moistening the selected absorbent point with the medication and then placing it in the canal, or by using a small syringe to place the medication in the canal and then inserting the absorbent point.

It is important that when an absorbent point is used in the canal, the point does not impinge upon the periodontal membrane. If this occurs, periapical irritation will result and the patient will experience considerable discomfort.

A small pledget of sterile absorbent cotton saturated with the medicament is then sealed into the pulp chamber of the tooth. The cotton pledget acts as a reservoir for the medication, and the point in the canal acts as a wick to carry the medicament throughout the root canal. It is believed that the tissue fluids which enter the canal from the periapical tissues will distribute the medication throughout the canal. It will then penetrate the dental tubules and thus destroy any remaining microorganisms.

In order to prevent the medication from escaping from the tooth, a double seal is generally employed. The inner seal, which is placed on top of the pledget of cotton in the pulp chamber, generally consists of gutta percha. After placing this, the excess medication is removed with a pledget of cotton saturated in alcohol or some other solvent.

An outer seal of some cement substance is then placed. This double seal technique is useful in preventing medicaments from escaping into the oral cavity, and also in preventing saliva with its contained bacteria from gaining entrance into the root canal.

In a study reported earlier by the author it was found that the root canal, including one-third of the pulp chamber, possessed a remarkably small volume.[13] In the central incisor this volume was approximately 0.02 cc. In the lower first molar there was approximately 0.05 cc. This included all three root canals completely enlarged, and one-third of the pulp chamber as well.

Since the absorbent point is made of relatively inert material and does not add to the antiseptic qualities of the root canal medicament, it was thought advisable to consider the use of medication in a paste form in order to obtain a larger volume of medication within the canal. This was expressed in a paper presented by the author when the D.C.P. was first introduced.[15] By utilizing the complete space which was present for medication alone, a greater volume could become available to destroy, or inhibit the growth of, any remaining microorganisms.

It was also found that when a paste material was sealed in the root canal, there was less tendency for periapical irritation. The possible explanation is that the absorbent point that is commonly used

in the root canal will expand when the tissue fluids enter the canal. This expansion causes pressure within the root canal, and is expressed by tenderness in the periapical region. These findings have been confirmed and reported by Wolfsohn.[16]

The method of inserting a paste material is as follows. The paste is carried to the root canal by means of either a syringe with a blunt needle or a metal instrument, depending upon its viscosity, and pumped into the canal from the pulp chamber. A small pledget of cotton is then placed in the canal, and gentle pressure exerted until the patient is aware of a slight amount of pressure in the periapical tissues. If this pressure is uncomfortable, the original pledget of cotton is discarded. This procedure will generally remove a sufficient quantity of medicament to eliminate the periapical discomfort when a second, and dry, pledget of cotton is applied.

The double seal is then used. As the tissue fluids now enter the root canal from the periapical region, the paste will be diluted and carried more freely throughout the root canal. In preparing root canal pastes, it is important to have a base that is water soluble so that it may be placed in solution by the periapical fluids, and may be readily removed from the canal when required.

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Obtaining and Maintaining Surgical Cleanliness

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The microorganisms present in the oral cavity can be divided into three groups. The first group comprises a large number of microorganisms that enter the oral cavity in the air and in food and water and that seldom are able to cause infection.

The second group consists of the truly pathogenic microorganisms, e.g., those that cause typhoid fever, diphtheria, scarlet fever, measles, mumps, chicken pox and poliomyelitis. These organisms are usually transient in the mouth, but in some of these diseases there is a carrier problem. The dentist who is careless about the disinfection of instruments and who allows organisms to be transferred about his office by the lack of such a simple procedure as handwashing between appointments may transfer virulent microorganisms between patients.

The third and predominant group of microorganisms is composed of those which are normally found in the oral cavity in large numbers and are classed as potentially pathogenic. These organisms may cause infection if the mucous membrane which acts as a barrier to infection is penetrated, if the oral tissues are traumatized, if organisms to which one patient has become immunized are transferred to a patient who is not immune to that particular strain, or if the patient's oral flora has become unstabilized by the use of antibiotics.

The importance of obtaining and maintaining surgical cleanliness has taken on a new aspect since the increase of infectious and serum hepatitis. These diseases are probably caused by two strains of the same virus. Both diseases, but particularly serum hepatitis, have increased markedly with the increased use of parenteral therapy. The diseases differ in that serum hepatitis has an incubation period of 80 to 160 days, during which the virus is present in the blood of the patient during the incubation period. Infectious hepatitis has an incubation period of 21 to 42 days. Serum hepatitis is transmitted solely by par-

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enteral introduction of the virus, and only 0.01 ml. of infected serum is necessary to transmit the disease. Infectious hepatitis may also be transmitted by the parenteral route but oral ingestion of fecally contaminated material is the more common means. Jaundice may be present in both types because of hepatic damage. Liver failure and death occur in both types, but the mortality rate is higher in serum hepatitis. Mild forms of both diseases occur and may not be recognized. The virus has been found in the blood of patients who have had serum hepatitis for as long as 5 years after the initial symptoms, that of infectious hepatitis for as long as 2 years.

Foley and Gutheim[6] report 15 cases of serum hepatitis, including 3 deaths, that were traced to injections made by dentists. The virus is unusually hardy and is resistant to chemicals. The World Health Organization Report on Hepatitis[4] states that "No chemical disinfectants are accepted for sterilization of instruments." Transmission results from inadequate disinfection of needles, syringes, cutting instruments, and equipment employed for injection and for capillary punctures, and from the use of multiple-dose syringes. The practice of reusing local anesthetics from vials in the cartridge-type syringe on different patients is to be condemned.

Surgical cleanliness may be achieved (1) by careful handwashing between patients and by care in handling equipment and instruments while working on a patient, to prevent microorganisms from being transferred to subsequent patients; (2) by preparation of the field of operation to decrease the number of microorganisms present and by the utmost care in handling tissues to keep trauma to a minimum; (3) by proper disinfection or sterilization and storage of instruments.

HANDWASHING AND HANDLING OF EQUIPMENT

While operating in the oral cavity, the dentist's hands are heavily contaminated by microorganisms from the patients' mouth. If these organisms are not removed they will be carried from patient to patient, from patient to equipment and instruments in storage cabinet and back to other patients, and from patient to dentist.

Handwashing is of prime importance to the dentist and to his patients. The use of detergents containing hexachlorophene markedly reduces the natural skin flora and eliminates contaminating organisms obtained from patient's saliva. Constant use of hexachlorophene leaves a water-insoluble film of this agent on the hands so that it has a continued effect. The dentist, after washing his hands, should take precautions not to recontaminate his hands by touching or handling unclean equipment or objects. To demonstrate the number of microorganisms on

the hands after washing with hexachlorophene soap (Fig. 1A) and microorganisms on the hands after making an oral examination (Fig. 1B) and touching some objects (Fig. 1C,D), the following procedures were carried out. Hands were rubbed on the surface of a blood plate, after which the plates were incubated. The growth is a partial indi-
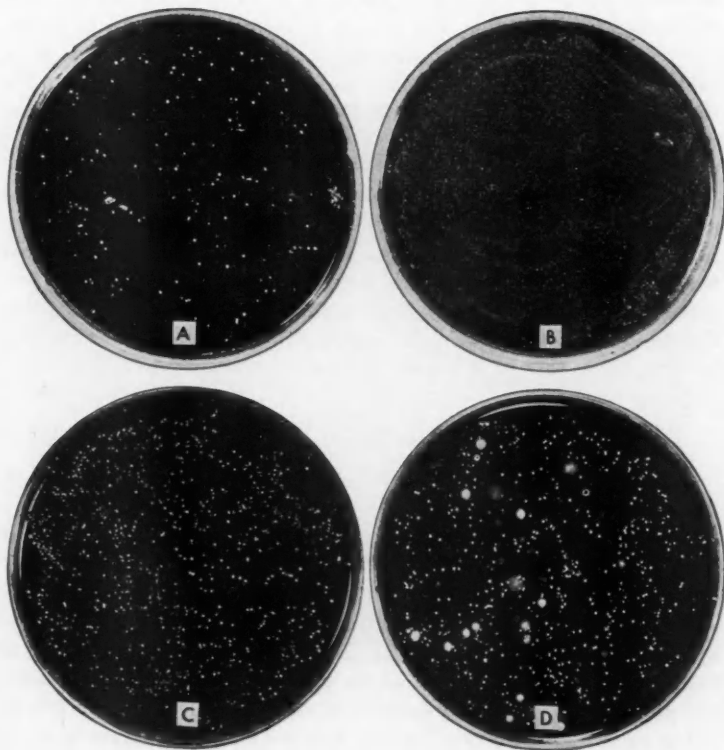
Fig. 1. Microorganisms on hands after: A, washing hands with hexachlorophene soap; B, making oral examination; C, touching faucet; D, handling telephone. (From Sommer, R. G., Ostrander, F. D., and Crowley, M. C.: Clinical Endodontics. Philadelphia, W. B. Saunders Co., 1956.)

cator of the number of microorganisms on the hands. The actual number present is much higher than represented on the plates, since viruses, spirochetes, some fungi, and the tubercle bacilli, to mention only a few, do not grow on this medium under the test conditions. The hands were clean, as in A, before each of the other procedures.

If the dentist's hands are contaminated, as they would be in the midst of operative procedures, instruments needed from the dental

cabinet may be removed from the cabinet by the use of forceps. A separate pair of forceps should be used for each patient. If placed on the operating table it may be kept within the folds of a clean towel so that it will not become contaminated by the patient's microorganisms. The light can be handled with a paper towel. The dentist should be aware of the possibility of cross infection in his office and should take every precaution to eliminate it. Unfortunately, many pieces of dental equipment cannot be disinfected properly. Some dentists clean chair arms, table tops, etc., with a disinfectant but the period of exposure to the disinfectant is too short to be effective. A good cleaning with soap and water will probably do more good since soap has slight disinfecting properties and, more important, is also a good mechanical cleanser.

Preparation of the operating field consists of wiping off the mucous membrane before applying a disinfectant, and then making injections or incisions of tissues. All microorganisms cannot be removed from the mucous membrane, but the preceding precaution will reduce the number of microorganisms introduced into the deeper tissues so that the tissues will be less likely to become infected. The less tissues are traumatized, the less chance there is for infection to occur.

STERILIZATION AND DISINFECTION

Sterilization is a process by which all forms of life are killed. A sterile instrument cannot carry microorganisms of any kind. *Disinfection*, however, is a process by which most microorganisms are rendered incapable of causing infection. This means that not all microorganisms can be killed by the disinfecting agent. Spores of spore-forming microorganisms are not killed by disinfecting procedures. In fact, the criterion for classifying an agent as a disinfecting or sterilizing agent is its action on spores. If spores which are usually resistant to chemical and physical agents are killed, then the agent may be classed as a sterilizing agent. The tubercle bacillus and the virus of infectious hepatitis and serum hepatitis are not susceptible to many common chemical disinfectants. A heavily contaminated object may also resist chemical disinfection because of the failure of the chemical to attack a wide variety of microorganisms. Protein material, such as blood and saliva, also reacts with chemical compounds so that penetration of the chemical to microorganisms is prevented.

Sterilizing Methods

Steam under Pressure. The autoclave is an apparatus in which steam under pressure can be raised to higher temperatures than can

free-flowing steam. No reliance should be placed on pressure gauges on autoclaves. Fifteen pounds pressure *should* raise the temperature of steam to 250° F. (121° C.), but a non-functioning gauge may read 15 pounds although the pressure may be much lower. Temperatures lower than 250° F. (121° C.) are not effective for sterilization. In using the autoclave certain precautions must be taken to ensure its maximum efficiency. It is the penetrating ability of steam which makes it an effective sterilizing agent. Because autoclaving does cause corrosion of instruments, many operators take all kinds of precaution
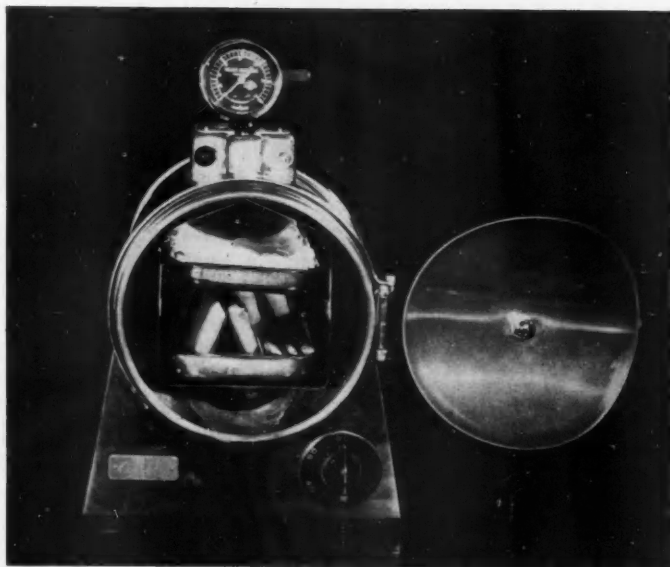
Fig. 2. Covered instruments on top tray, and paper-wrapped towels, sponges, and swabs on lower tray—arranged to let steam penetrate packages easily.

to protect instruments from the action of steam. While these measures do protect instruments from corrosion, they interfere with the sterilization process. Packaging of instruments must be such that the steam can penetrate fully to all parts of the contained instruments. Paper and cloth will serve the purpose, but metal containers and plastics will not allow penetration of the steam. The autoclave and objects in it will be filled with air, which must be evacuated and replaced with steam. Towels, sponges, etc., must not be packed so tightly in the autoclave that the steam cannot penetrate them. Figure 2 shows the proper arrangement of instruments and packages in autoclave. A common practice in dental offices is to protect injection needles by insert-

ing them in cotton rolls—this hinders the penetration of the steam to the lumen of the needles and inadequate sterilization results.

We have attempted to overcome the corrosive effect of moisture on metal instruments by coating them with an oil emulsion before autoclaving. The emulsion does not prevent the penetration of steam, but does protect metal from its corrosive action. It also provides enough lubrication for hinged instruments. Handpieces may be autoclaved after coating with emulsion and relubricating after sterilization. This method, still in the experimental stage, may be used for all instruments except root canal files and reamers and injection needles and syringes. At this time the most effective emulsion is made up of

    5% Finoil (a light mineral oil)
    1.25% Span 80 (sorbitan oleate)
    1.25% Tween 80 (polyoxyethylene [20] sorbitan mono-oleate)
        Water

Alcohol mixtures heated to a vapor in a device similar to an autoclave will not corrode instruments, but neither will they sterilize.

Dry heat sterilization is as effective as autoclaving, but it has two distinct disadvantages. These are the longer length of time necessary to achieve sterilization and the fact that any material injured by heat cannot be sterilized in this way. A temperature of 320° F. (160° C.) is the minimum that may be used. The time of exposure varies from 1 to 2 hours, depending on the load being sterilized. When only a few instruments are being sterilized in the oven, 1 hour may be sufficient, but if the oven has a capacity load, a minimum of 2 hours is necessary. The time factor is too great for the use of this method in the dental office where many instruments must be reused frequently. The method is best suited to root canal instruments where rusting due to exposure to moisture is a serious problem, or to injection needles, towels, sponges, etc., where it may be used as a second choice to autoclaving.

Disinfecting Methods

Since disinfecting methods are not capable of killing all microorganisms, instruments should be thoroughly cleansed before any disinfecting process. Blood and saliva on instruments will interfere with the action of the disinfecting agent. Rinsing in cold water followed by scrubbing with soap and water will mechanically remove blood, saliva, and microorganisms from the instruments and insure a higher degree of success whatever disinfecting method is used.

Boiling Water. Boiling in water for 10 minutes is the most efficient and satisfactory method of disinfecting instruments. Some authorities suggest a 20 minute period of boiling, claiming that spores are killed

within this time, but McCulloch[9] reports that some spores are resistant to 3 hours of boiling. Since one should not expect to kill spores by boiling, a 20 minute period is not necessary. The addition of an alkali to the boiling water will increase its bactericidal action.[2,9] Even the virus responsible for infectious hepatitis is susceptible to the action of the alkalies and boiling water.

Because metal tends to rust and dull when boiled, boiling is not used for expensive instruments. However, for cheaper, easily replaced instruments and those not injured by boiling, it should be the disinfecting method of choice. The harmful effects of boiling may be reduced by (1) using distilled water, or tap water that has been previously boiled; (2) adding an alkali such as tri-sodium phosphate (1 per cent), sodium carbonate or borax to the water; (3) cleaning boilers frequently to eliminate scale deposits. The occasional addition of acetic acid or vinegar to the water in the container and boiling for about 10 minutes will help to remove scale. The container may then be emptied and the scale removed easily. Instruments should not be left in the boiler during this process; (4) removing instruments from the water while they are still hot and drying them immediately. The practice of allowing instruments to cool in water after boiling is one of the commonest causes of rusting.

Chemical Disinfectants. This is the most unreliable of the common methods of disinfecting. It should be used only for instruments that because of cost cannot be boiled or sterilized. The importance of thoroughly scrubbing instruments before chemical disinfection cannot be overemphasized. Blood and saliva, if left on instruments, will prevent contact between microorganisms and the chemical solution. It is also important that instruments be dried after rinsing so that no moisture will be left on them to dilute the disinfecting chemical.

Bacteria from the air and hands may also be deposited in the chemical solution, thus rendering it less capable of reacting with the bacteria on instruments. Solutions should always be kept covered and instruments should be placed in and removed from the solution with forceps. Solutions should be changed frequently.

Injection needles should be sterilized in the autoclave or oven or disinfected by boiling for 10 minutes, after thorough rinsing. If the cartridge-type syringe is used for procaine injection, the unused portion in the cartridge should be discarded because blood may be drawn into the cartridge during the injection.

The number of chemical disinfectants available is legion, and for some, at least, absurd claims have been made as to their efficiency. The dentist is best guided in his choice by selecting from those accepted by the Council on Dental Therapeutics. The period of ex-

posure of the instruments to the disinfecting solution should be 30 minutes. This is a longer time than is recomended by manufacturers, who usually suggest the minimum time necessary. The longer time provides more safety. Some of the commonly used disinfectants are:

Quaternary Ammonium Compounds. Many of the proprietary disinfecting solutions now on the market belong to this class. Washed and dried instruments should be exposed to the solution for 30 minutes. Soap will inactivate the quaternary ammonium compounds, so instruments should be thoroughly rinsed after washing. Rubber is softened by these compounds.

TABLE 1. SUGGESTED TEMPERATURES AND TIMES FOR MOLTEN METAL AND BEAD STERILIZERS

AUTHOR	TEMPERATURE	TIME		
		FILES	PAPER POINTS	COTTON PELLETS
Boak[1]	Not given	5 sec.	10 sec.	Not done
Stewart and Williams[12]	218° C. (424° F.) to 280° C. (536° F.)	2 sec.	5 sec.	10 sec.
Grossman[7]	204° C. (400° F.)	5 sec.	10 sec.	10 sec.
Grossman[8]	225° C. (436° F.)	5 sec.	5 sec.	5 sec.
Findlay[6]	271° C. (520° F.)	9 sec.	17 sec. Charring	24 sec. Charring

Nitromersol N.F., a mercury based compound, cannot be used for aluminum instruments. The solution is highly alkaline and may be disagreeable to the operator and the patient if instruments are not rinsed in water before using.

Formaldehyde germicides have a highly objectionable odor, and contact may lead to dermatitis in hypersensitive persons.

Alcohol, 70 per cent, is not a good disinfecting solution.[2]

Phenol and cresol compounds are often injurious to instruments.

Molten Metal and Glass Bead "Sterilizer." The molten metal sterilizer is a low-fusion alloy which melts and can be raised to high temperatures in an electrically controlled container. The device has been used mainly for root canal instruments. Since the molten metal sticks to instruments, paper points and cotton pellets, the metal has been replaced with very small glass beads less than 1 mm. in diameter.

Table 1 shows the times and temperatures suggested for molten metal and bead sterilizers. Since there is some question as to the ability of this device to kill spore-formers on small instruments in a

reasonable length of time, it would be better to use other methods of sterilization for root canal instruments. It cannot be used for large instruments.

Flaming. An open flame such as a Bunsen burner or an alcohol lamp may be used for flaming metal instruments. However, the instruments must be heated thoroughly so that microorganisms are burned off. This, of course, affects the temper of the metal. Flaming is most useful for forceps, which have been previously disinfected, immediately before removing instruments from the root canal tray. The forceps should be thoroughly heated, not merely passed quickly through a flame. This method is not recommended for files and reamers.

Hot Oil. Mineral oil, silicones and other synthetic oils have been used for hot disinfection of handpieces and jointed instruments that are dulled or rusted by moisture. The instruments should be cleansed in a commercial cleanser such as Stoddard's Solvent and immersed in oil in an oil "sterilizer." An exposure to 150° C. (300° F.) for 10 minutes or 121° C. (250° F.) for 15 minutes will kill all microorganisms except spore-formers. These will be killed at a temperature of 150° C. (300° F.) in 1½ hours' exposure; if the latter temperature and time are used, oil may be an effective sterilizing agent. No oil now in use is completely effective as both a lubricant and a disinfectant. Some of the oils, such as the hydrocarbon oils, are good lubricants but on heating to the desired temperature they break down and give a disagreeable odor. The critical temperatures for killing vegetative microorganisms cannot be lowered without considerable prolongation of the heating time, so the use of these oils is impractical. Furthermore, the hydrocarbons have such a low flash point that they cannot be heated much above 250° C. without danger of ignition.

Silicone oils, while they have a high flash point and can be heated without producing disagreeable odors, are not very good lubricants. For this reason, they are not as effective for handpieces but are effective for hinged and jointed instruments.

In interpreting suggested temperatures and times for using hot oil, it should be kept in mind that it is only the heat produced which is effective in disinfecting so that comparisons to autoclaving or boiling are misleading. The temperature and time for hot air sterilization or disinfection are more readily comparable.

INSTRUMENT STORAGE

Sterilized instruments are not difficult to store, since instruments, towels, sponges, etc., may be wrapped in such a way before sterilization in autoclave or oven that they will remain sterile until ready for

use, if it is necessary to maintain sterility. This applies particularly to injection needles and syringes, where there is no reason why they should be exposed to air after sterilization until ready for use.

Disinfected instruments should be placed in clean closed cabinets, immediately after they have been dried. The storing of autoclaved or boiled needles and/or syringes in chemical disinfectants is not a good procedure. Tetanus, serum hepatitis and pyocyaneus infections have been traced to this practice. In all cases the microorganisms causing these infections were traced to the contaminated disinfecting solution in which the needles had been stored *after* adequate autoclaving or boiling. Boiled needles should be kept in the folds of *clean*, dry towels (if sterile towels are not available); if autoclaved or dry heat sterilized, they are stored in the containers or the wrappings in which they were sterilized.

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Bacteriology in Endodontic Treatment

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In the practice of root canal therapy, the efforts of the practitioner are directed at the elimination of infective microorganisms, the maintenance of asepsis and the restoration of tissues to optimal health and function to resist infection. Bacteriologic examination is indispensable in the practice of endodontia and provides (1) information on the bacteriologic status of the pulp canal, apical and periapical tissues; (2) control over aseptic techniques (saliva and seal leakage, instrument contamination, presence of carious dentin, etc.); (3) differential diagnostic information on the nature and origin of inflammatory reactions in endodontal tissues; and (4) standard methods for the isolation, identification and assay of microorganisms for their sensitivity to antimicrobial agents.

The basic inflammatory reaction is the same in the pulp, periodontal tissues and bone whether it results from streptococci or cresol, and the cardinal symptoms of the inflammatory reaction apply. The irritant initiating the inflammatory reaction might be thermal, chemical, traumatic, or microbial in origin. It expresses itself clinically in pain, swelling and roentgenographic changes in the supporting bone. An anachoretic effect, or tendency of microorganisms to become fixed in areas of inflammation, has been well established, and the appearance and persistence of an exudate is one of the most important evidences of an infective inflammatory reaction.

Bacteriologic examination of this exudate, whether of pulpal, apical or periapical origin, is significant because (1) there are no characteristic periapical roentgenologic changes associated with the presence of infection or with any specific bacterial forms; (2) no particular organisms can be associated with a specific type of pulp involvement or periapical disturbance; (3) odor or lack of odor as a criterion of sterility is futile—few of the cases of pulp-involved teeth show

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proteolytic microorganisms; (4) a clinically and roentgenographically satisfactory result is more probable when a canal is filled only after a negative culture than when the canal is filled in spite of positive culture; (5) in three separate studies, the clinician's "opinion" that the tooth was ready for filling was not confirmed over 40 per cent of the time by bacteriologic cultural findings; and (6) cultural procedures provide some saving of time because most dentists who do not use cultures tend to treat teeth for a longer period of time.

Whereas the presence of microorganisms does not prove infection, their ability to invade and multiply in the tissues of the host does. As a result of altered environmental conditions or the diminished resistance of the tissues of the host, these microorganisms become virulent. Their importance should not be minimized nor their numbers discounted. A clinically and roentgenographically symptomless pulp-involved tooth with bacterial contamination can manifest an acute infective pulpitis or apical periodontitis within hours, owing to mechanical, chemical or physical factors.

An infected root canal may be visualized as a "zone of infection" characterized by the products of suppuration—bacteria, pus, necrotic tissue and their products. A variable area in the vicinity of the apical foramen is the "zone of contamination" wherein the products of suppuration are provoking either stimulative processes of repair or cellular destruction. Usually both are manifest. The acute periapical abscess represents an extension of the zone of infection to apical and periapical tissues; the chronic abscess, the granuloma and the cyst are the manifold manifestations of cellular stimulation and destruction occurring simultaneously in the tissues in response to the irritant.

BACTERIOLOGY OF THE NORMAL AND THE PULP-INVOLVED TOOTH

The Normal Tooth

Early investigators utilizing the extraction method for obtaining cultural material for bacteriologic appraisal consistently reported the finding of microorganisms in the pulpal tissue and periapical region of normal teeth. However, careful studies by many investigators have since shown that it is practically impossible to remove a tooth aseptically. The high percentage of positive cultures must be considered in a large measure the result of contamination, not infection; the source of the microorganisms, at least in extracted teeth, is presumably the highly contaminated gingival crevice. Furthermore, smears and sections from such teeth show no evidence of infection. The pulps and periapical tissue of vital healthy teeth are invariably free of micro-

organisms except during periods of bacteremia, either transient or sequent to systemic disease.

The Pulp-involved Tooth

Bacteria or their products may gain access to the dental pulp via (1) accidental exposure or carious extension, (2) the periodontal tissues, (3) the blood stream, and (4) by extension from a periapically involved adjacent tooth.

Over 80 per cent of pulp involvements result from carious exposure. Many factors contribute to the rapidity of the onset of clinical symptoms. Prominent among these is the resistance of the tissues of the host; the nature and numbers of the contaminating microbial population; the degree and persistence of salivary contamination; the physicochemical environment; and the adequacy of coronal drainage for the products of microbial metabolism and tissue response.

The ability of a microorganism to infect tissues is associated with its capacity to produce one or more virulence factors, viz.: alpha and beta hemolysins, fibrinolysin, coagulase, hyaluronidase and collagenases. The production of these by organisms isolated from the saliva and pulp-involved teeth has been amply demonstrated.

The work of many investigators suggests that the irritation of the pulp from cavity preparation, the placing of filling materials, the use of sterilizing drugs, heat, cold and other stimuli may be capable of attracting and fixing bacteria present in the blood stream during transient bacteremia. Cases of postoperative "idiopathic" pulpitis may be a result of anachoresis. The course of events is summarized by Robinson and Boling[9] as: (1) operative or postoperative irritation of the pulp with the production of asymptomatic pulpitis, (2) subsequent development of transient bacteremia, and (3) anachoresis with infection of the pulp. Macdonald et al.[6] noted that the mere presence in the pulp of bacteria, even known pathogens, does not necessarily lead to disease. It seems safe to conclude that the presence of bacteria in pulps already inflamed might compound the injury and lead to necrosis.

The bacteriologic status of the traumatically induced non-vital pulp has been studied by a number of investigators. Stewart[12] reported on 14 devitalized teeth in which organisms were recovered from all 14. Macdonald[6] reported that 38 of 46 such teeth yielded growth which was representative of the oral microbiota. Thirty-two of the 71 strains isolated were anaerobes, and it was postulated that most of the organisms reached the pulp from the oral cavity via the lymphatics and blood vessels of the periodontium. The variety of microorganisms iso-

lated by Smith[11] in a study of 60 unexposed root canals of non-vital periapically involved teeth appears to be of oral origin and lends support to the probable extension of these strains from the periodontal tissues.

Microorganisms Isolated from Pulp-involved Teeth. The oral resident and transient microbiota is inherently a varied, complex and heterogenous population. Their nutritive and physico-chemical requirements differ markedly and certainly no currently available bacteriologic medium can promote and sustain the growth of many of the members of this population. At best our standard procedures provide only relative information, and frequency tables depicting the species of microorganisms from pulp-involved teeth in qualitative and quantitative relationships are incomplete. Overemphasis is placed on the more readily cultured organisms. Often the morphologic forms predominant in microscopic studies are not cultivable. These morphologic types include filamentous forms, fusiforms, vibrios, spirochetes, pleomorphic bacilli, etc.

Variations in the method of obtaining the culture as noted previously, in the time of culturing and in the culture media utilized in studying the bacteriology of the pulp-involved tooth have resulted in considerable disagreement among individual investigators. Since such a diversity of findings and techniques exists, only broad conclusions may be made. The microorganisms isolated from pulp canals and periapical areas and those found in the oral cavity appear to be the same. The isolation of *Salmonella typhosa, Mycobacterium tuberculosis* and *M. leprae* have been reported from the pulps and periapical granulomas in cases with systemic disease.

A considerable number of negative cultures are obtained on the initial opening of the root canal. Data reported by numerous investigators support the contention that between 30 and 40 per cent of pulp-involved teeth are initially sterile. Grossman[3] recently reported on 771 pulps cultured from cases of pulpitis. Approximately 50 per cent showed positive coronal and radicular cultures; 25 per cent showed negative coronal and radicular cultures; 8 to 9 per cent gave positive coronal cultures and negative radicular cultures; and 15 per cent gave negative coronal cultures and positive radicular cultures. Hedman[4] reported on 82 cases of pulp-involved anterior teeth which had radiolucent areas and which were cultured by the cannula–culture wire method. Fifty-six, or 68.5 per cent, had viable bacteria in both the pulp canals and periapical areas; 8.5 per cent had bacteria in the pulp canal but not in the periapical areas; and 23 per cent were negative throughout. All of the patients having streptococci in the pulp canals had streptococci in the periapical tissues.

The organisms isolated most frequently from root canals are the alpha hemolytic (viridans) streptococcus, *Staphylococcus albus* and the gamma (non-hemolytic) streptococcus. As may be noted in Table 1, over 80 per cent of a series of cultures in two separate studies contained streptococci.

Cran[1] has reported that many of the *Staphylococcus aureus* strains isolated were coagulase positive and therefore might be considered potential pathogens.

In addition to predominant species heretofore mentioned, over 30 other species and varieties have been reported. Included in the group

TABLE 1. *Prevalence of Streptococci in Root Canal Cultures*

NO. OF POSITIVE CULTURES	NUMBER OF CULTURES CONTAINING STREPTOCOCCI			NUMBER OF CULTURES NEGATIVE FOR STREP
	Pure Culture	Mixed Culture	Total	
*357	191 (53%)	103 (29%)	294 (82%)	63 (18%)
†256 vital	101 (39.4%)	107 (45.4%)	208 (86.3%)	48 (13.7%)
165 non-vital	52 (32.1%)	76 (46.0%)	129 (78.1%)	37 (22%)

* Sommer, R. F., Ostrander, F. D., and Crowley, M. C.: Clinical Endodontics Philadelphia, W. B. Saunders Co., 1956, p. 398.
† Author, unpublished data.

are members of the following genera: Fusobacterium, Lactobacillus, Corynebacterium, Diplococcus, Neisseria, Veillonella, Actinomyces, Bacillus, Micrococcus, Escherichia, Aerobacter, Pseudomonas, Hemophilus, Treponema, Streptococcus, Monilia, Cryptococcus, Saccharomyces, Clostridium, Sarcina, Rhodotorula, Streptomyces and Paracolon bacilli.

The numbers and significance of anaerobes in pulpal and periapical tissues have received comparatively little attention. Crowley[2] and Shay[10] suggest that strict anaerobic methods are not necessary for the cultivation of microorganisms from root canals. Morse and Yates,[8] however, reported 43 positive anaerobic cultures among 153 teeth cultured. Cran[1] reported the isolation of anaerobic streptococci in 18.7 per cent of total occurrences of microorganisms. Leavitt et al.[5] found that 5 of 35 cases cultivated anaerobically yielded chiefly streptococci after two consecutive negative cultures by aerobic methods.

The oxygen requirements of microorganisms isolated from pulpless

teeth have been carefully studied and reported by Mazzarella et al.[7] as follows: facultative anaerobes, 45 (50 per cent); anaerobes, 22 (24.4 per cent); aerobes, 22 (24.4 per cent); 10 per cent carbon dioxide, 1 (1.1 per cent).

The question whether rendering the root canal sterile accomplishes sterilization of periapical tissues has been a matter of conjecture. Hedman's findings indicate that in anterior teeth with open foramina, "after two successive negative cultures were obtained in the treatment of the pulp canal, no growth was obtained from the periapical tissues of any of the 56 patients studied."[4]

BACTERIOLOGIC PROCEDURES IN CASE MANAGEMENT

A direct smear made from pulpal tissues or fluid exudate provides diagnostic information on the nature and numbers of the cellular elements present. The presence of increased numbers of polymorphonuclear neutrophiles characterizes an acute inflammatory process, and increased numbers of macrophages, lymphocytes and plasma cells are present in a chronic inflammatory reaction. In the absence of a cellular response, one should regard the presence of microbial morphologic types as contaminants.

The morphologic appearance and staining reaction of the various bacterial forms noted in these smears provide presumptive diagnostic information and indicate cultural methods. It should be noted that the direct smear represents the total microbial population, viable and non-viable. When only non-viable organisms are present, a negative culture will obtain. Many thousands of microorganisms must be present in a specimen before they may be visualized by optical microscopy, and yet cultural techniques will yield positive findings. The direct smear and culture serve to complement each other.

The Culture Medium

A number of media for culturing root canals have been recommended: brain heart infusion, trypticase dextrose broth, Brewer's thioglycollate, Robertson's cooked meat medium, and trypticase soy broth. Some investigators have added dextrose to these media to promote the growth of acidogenic microorganisms; ascitic fluid in 5 per cent concentration to promote the growth of fastidious microorganisms; and agar in 0.1 to 0.3 per cent concentration to permit varying levels of oxygen tension. It should be understood that no single medium can support the growth of all of the bacterial flora of the root canal. When the special growth requirements and complex relationships of

the mixed bacterial oral population are better understood, more precise cultural methods and media will become available.

The use of a trypticase soy broth (B.B.L., No. 01-162) with 0.2 per cent agar appears to support and permit the isolation of much of the aerobic, anaerobic, and microaerophilic microbiota isolated from pulp canals. When penicillin is being utilized in chemotherapy, the addition of 1 ml. of penicillinase (B.B.L., No. 02-407) to each 500 to 1000 ml. of medium will serve to inactivate penicillin. The medium is best stored in screw-top test tubes at room temperature without agitation. No effective neutralizer for antibiotics such as streptomycin, bacitracin and the tetracyclines is available, and one must depend on dilution to minimize their antibacterial effects. False negative cultures may occur when antibiotics are being used during therapy.

Cultures should be incubated for a minimum of 48 hours at 37.5° C. (98.6° F). Suitable inexpensive incubators may be obtained from local laboratory supply houses. A tightly insulated box with a 40 watt bulb as a heat source and a chicken brooder type thermoregulator will serve adequately.

Taking the Culture

A culture should be taken at each of the last two visits prior to filling the root canal. The following steps are utilized in obtaining these cultures: (1) isolate tooth under rubber dam; (2) sterilize tooth surface with suitable bactericidal agent, such as tincture of iodine, thimerosal or benzalkonium chloride; (3) with *sterile* instruments remove dressing and irrigate canal with hot *sterile* saline or water; (4) absorb irrigant with *sterile* paper points until the last point is wet for a distance of 1 mm.; (5) allow the next paper point to remain in the canal for its entire length for a period of 1 to 2 minutes; (6) remove this point and insert into tube of culture medium. Be sure to flame the orifice of culture tube on opening tube and prior to replacing the screw cap.

Interpreting the Cultural Result

In the absence of bacterial growth, the culture medium will remain clear. A negative culture obtains when (1) the canal is sterile, (2) too few microorganisms are present to initiate growth, (3) sampling is inadequate, (4) an inhibiting concentration of antimicrobial agent has been carried over, and (5) the medium will not support the growth of the more fastidious microorganisms. False negative cultures may occur following the use of antibiotics in endodontic treatment.

Aside from this situation, it is reasonably safe to assume that if our less culturally fastidious microorganisms have been eliminated, all microbial life has been eliminated.

A positive culture will show some degree of turbidity or flocculence (Fig. 1). It may vary from a tenacious mucoid mass associated with the tip of the cotton point to a fine, evenly dispersed granular growth. A surface pellicle or growth at the top of the paper point where con-
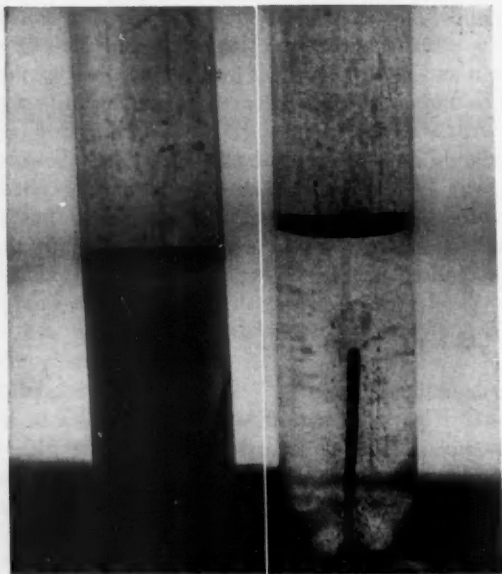
Fig. 1. *Left,* Positive culture. *Right,* Negative culture.

tact was made with forceps suggests contamination. Neither the degree nor the character of the growth is necessarily indicative of the types or numbers of microorganisms in the root canal. Subcultures on suitable media will provide a means of identifying species, and pure cultures of these isolates may be then tested for drug sensitivity.

The microbial culture is streaked heavily on the surface of the culture medium. Four to six paper points saturated with antibacterial agents are then placed on the surface and the plates are incubated at 37.5° C. for 24 to 48 hours. Zones of inhibition give relative information of microbial sensitivity (Fig. 2).

Positive cultures will be obtained as a result of frank infection or contamination of the aseptic field by saliva or unsterile instruments. In either case, the filling of a root canal in the face of a positive culture
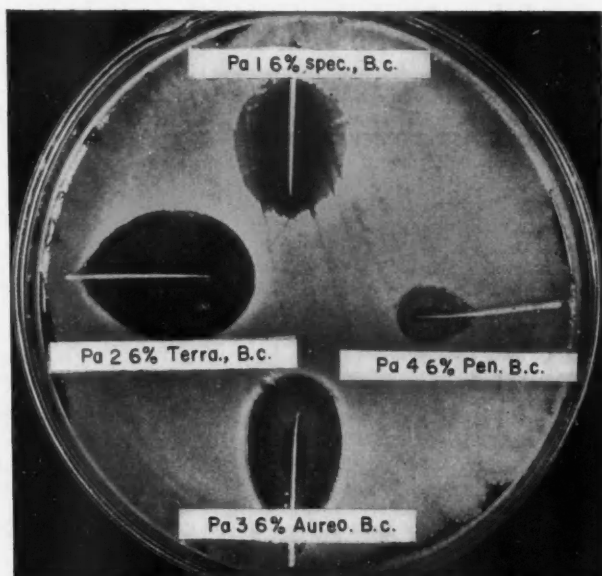
Fig. 2. Zones of inhibition indicating relative sensitivity of *Bacillus cereus* to 6 per cent aqueous solutions of penicillin, Aureomycin, Terramycin, and Spectrocin (neomycin-gramicidin).

is contraindicated. Sound endodontic practice should provide for bacteriologic control in case management.

SUMMARY

The successful retention of the treated pulpless tooth as a healthy functioning member of the dental arch is an outstanding achievement. The application of bacteriologic methods to endodontic practice complements sound clinical techniques and judgment. It serves as a continuous control and a measure of aseptic technique, efficacy of drug treatment and surgical débridement. It restores confidence in the practitioner by providing objective evidence to substantiate his skill and competence.

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The Obturation of the Root Canal

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The obturator is the material or combination of materials used to effect closure of the pulp canal and to produce the hermetic seal.

A hermetic seal is a seal that prevents the passage of air. When this term is utilized in association with endodontology, it also implies the complete filling of a root canal in its entirety and in all dimensions.

PREREQUISITES FOR OBTURATION

1. The canal must be properly prepared to receive the filling material. The preparation includes the enlargement of the canal to a size which will permit the free introduction of filling material into any aspect of the canal without obstruction. Enlargement to an ideal size also suggests instrumentation until all debris has been removed and irregularities such as nodules, fissures, and constrictions have been filed away. Upon completion of instrumentation, the root canal should be essentially in the shape of a cone with the apex just at the orifice of the apical foramen and the base at the opening into the tooth.

2. Prior to the permanent placement of the obturator, two successive negative cultures are required, each culture having been incubated for a minimum of 72 hours.

3. The tooth should be comfortable to forces that are within normal limits (no acute pericementitis).

4. The canal should be relatively dry or it should be capable of being dried with ease.

5. When a fistula is present prior to treatment or occurs during the course of therapy, the pathologic orifice must have completely disappeared and the surface continuity must have been established before obturation is permissible.

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6. The canal must be free of foreign and putrescent odors. When canals have been imperfectly instrumented or when accessory canals are present and undetected, tissue decomposition does take place. The products of tissue decomposition act as toxins that may be as injurious to periapical tissues as bacteria. This situation may explain some endodontic failures that occur in spite of the fact that negative cultures have been obtained.

THE GOAL OF OBTURATION

Once the canal or canals of a tooth have been rendered sterile, there may be some question as to the necessity for obturation. Available evidence demonstrates that an unfilled canal or a canal that is not hermetically sealed will act as a site where tissue exudates may accumulate and stagnate. Protein decomposition products act as toxins to tissues adjacent to the periapical lesion and prevent healing. The periapical lesion will be maintained by the continuous presence of toxins and may be subjected to subsequent secondary infection by way of hematogenous routes. The purpose of obturation becomes clear; the process should hermetically seal the root canal in all its dimensions. The obturator must be stable both physically and chemically, and it should not absorb fluids.

THE APICAL EXTENT OF THE FILLING MATERIAL

Opinions vary as to the ideal extent or location of the apical end of the obturator. It might be desirable to fill a root canal at a level even with the apical foramen, but this is done so infrequently as to make it a rarity. Examination of the actual border of the orifice of an apical foramen grossly, microscopically or roentgenographically discloses that no aspect of the orifice is in the same plane with any other aspect of the orifice (Figs. 1 and 2). Since this is true, placement of the obturator so that it is flush with the mesial aspect of the foramen without contouring it is likely to overfill or underfill the distal aspect, or vice versa. If the obturator is placed so that it is just at the labial aspect of the orifice, the filling material will, in all probability, be long or short at the lingual aspect. Since there is no method of obtaining a roentgenographic image from labial to lingual, contouring the obturator in this dimension is impossible. Thus, filling a root canal to the apical foramen with no portion of the obturator long or short is for all practical purposes impossible. There is sound biologic evidence to suggest that it is also undesirable. A point that is frequently overlooked is that minute dimensional changes usually occur at the apical
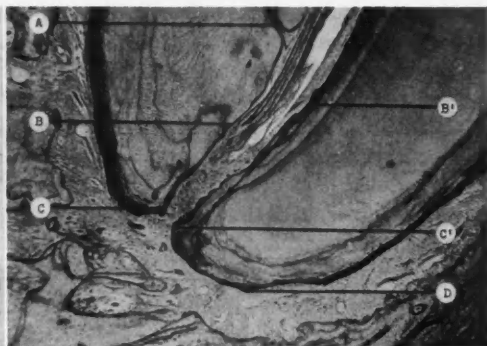
Fig. 1.   Fig. 2.

Fig. 1. Low power photomicrograph of mandibular incisor demonstrating relationship of apical foramen to long axis of tooth and to actual length of root. Note that apex of root and position of apical foramen are not at same level. *A*, Lingual aspect of apical foramen; *B*, labial aspect of apical foramen; *C*, apex of root.

Fig. 2. Periapical area of tooth in Figure 1. *A*, Accessory canal; *B*, dentino-cemental junction on lingual wall of root canal; *B'*, dentino-cemental junction on labial wall of root canal; *C*, lingual aspect of apical foramen; *C'*, labial aspect of apical foramen; *D*, apex of root.

Fig. 3. *A*, Roentgenogram made at appointment when endodontic therapy was undertaken. *B*, Roentgenogram made at completion of obturation; note position of arrow, and that root canal is slightly underfilled. *C*, Roentgenogram made one year following completion of therapy. Note resolution of area of periapical radiolucency, and position of arrow. As a result of a slight amount of root resorption, the obturator is now long or overextended.

end of the root during the resolution of an inflammatory process or the healing of any area of gross periapical pathosis (Fig. 3). If the periodontal membrane is intact and there is inflammation only in the periapical portion of this membrane, the irritation and the increase in vascularity may stimulate the resorption of cementum and the process will continue in varying degrees until the periodontal membrane re-

establishes normal arrangement and function. When periapical pathology has destroyed the apical portion of the periodontal membrane, the denuded cementum commonly is resorbed. After the periodontal membrane has been re-established, new cementum is formed covering the defect and frequently the apical foramen. Thus root resorption occurs in varying degrees where there is inflammation of the periodontal membrane and destruction of large areas of periapical tissue.

When a root canal is filled to the apical foramen in a tooth in which resorption is likely to continue during the healing process, it is possible that such a tooth ultimately may be overfilled (Fig. 3). Healed and newly formed periodontal membrane can and does de-

Fig. 4. Roentgenogram of a poorly obturated root canal. The arrows in the periapical area outline the radiolucent area surrounding the overextended obturator. The arrows directed toward the root canal denote absence of lateral condensation.

posit secondary cementum so that resorbed cementum may be replaced in varying degrees. As for overfilling a root canal, there is never justification for this practice even when the filling material is resorbable. No matter how stable, inert, or tissue-tolerated the filling material is, contact with it by the tissues will produce a foreign body reaction. Since the tooth in function is constantly being moved in the alveolus, the projecting filling material mechanically irritates the intact tissues or the tissues that are in the process of reestablishing normal anatomic form. An overfilled canal does not necessarily assure a well-condensed filling, and the overfilling is always an irritant that should be carefully avoided (Fig. 4).

In the procedure of choice, the root canal should routinely be obturated just short of the shortest length of the canal. The canal should be filled so that the proliferating periodontal membrane will at first

project into the unfilled portion and then ultimately lay down secondary cementum to produce the final seal of the canal. When the space between all aspects of the actual apical orifice and the apical extent of the obturator is from 0.5 to 1 mm., there will be a free physical and chemical interchange between the fluids in the unfilled portion of the canal and the fluids around the apical end of the root, and consequently no stagnation.

REQUIREMENTS FOR A FILLING MATERIAL

1. A good filling material should have the property of plasticity, permitting complete lateral and vertical filling of the root canal.

2. The filling material should be sterile. Sterility can be maintained by storage in an antiseptic solution.

3. The filling material should not be brittle or friable nor should it be excessively malleable.

4. It should be easy to place into a root canal and just as easy to withdraw. It should be possible to repeat the procedure for as many times as necessary to secure the correct dimensions.

5. It is preferable for the filling material to be stable and inert with no antiseptic or germicidal properties, but it should not encourage the growth of bacteria.

6. The material should certainly not shrink and preferably should not change in volume after insertion.

7. It should not absorb fluids or moisture.

8. It should be non-irritating and have a high degree of tissue compatibility.

9. It should not discolor the tooth.

10. It should be a poor conductor of heat.

11. It should be radiopaque. This characteristic will facilitate the detection of the filling and will permit evaluation of the thoroughness with which it obliterates the pulp canal.

12. The obturating material should be relatively easy to remove from the canal either in whole or in part. Complete removal will permit re-treatment. Removal in part will enable the use of a post or dowel.

METHODS OF OBTURATING A ROOT CANAL

Gutta Percha Method

Sectional Method. Once the canal has been correctly instrumented, fit a gutta percha cone to the canal. Make adjustments in the cone until

it is the correct length and until it is as perfectly adapted to the walls of the canal as possible. When this phase is completed, try gutta percha pluggers into the canal until one is selected that can be placed within 3 or 4 mm. of the apex of the root. In this position, the plugger should bind against the wall of the canal. At this stage, make a roentgenographic examination with the plugger in place. This will reveal the relation of the tip of the plugger to the apical orifice.

Before removing the plugger, place a marker on the shaft even with the incisal or occlusal surface of the tooth being treated. After securing the marker, remove the plugger. Section the previously contoured gutta percha cone with a sharp lancet into pieces 3 or 4 mm. in length, taking care to keep the sections in order. Then carefully coat the walls of the canal with root canal sealer. By warming the plugger, the apical section of the gutta percha cone can be fixed to its tip and then carefully carried to the root canal. Move the plugger gently and slowly into the canal so as not to force any of the sealer through the apical foramen or trap air in the canal. When the marker on the plugger shaft is even with the incisal or occlusal surface, move the plugger back and forth in an arc so that the gutta percha section will be fixed in the canal and detached from the plugger tip.

Now take a roentgenogram to determine the position of the inserted section. If it is short of the apex of the canal, select a slightly smaller plugger, place a marker on the shaft at the correct distance, warm the plugger tip, and work the apical section of gutta percha into the proper position. This section should be roentgenographically examined after each instrumentation until it is ideally positioned.

After closure of the apical foramen, add additional sections of gutta percha to the canal until it is completely filled. Pack each successive section of filling material firmly against the previous section so as to produce a homogeneous mass and to drive the sealer cement into the minute irregularities of the canal. Remove the portion of material projecting coronally to the cervical line with a warm plastic instrument. If the apical portion of the canal is wide, and there is the possibility of displacing the first section of gutta percha, the operator may elect to finish filling the canal at a subsequent visit, thus allowing the cement to harden and the first section of filling to become firmly fixed.[3]

There are several problems inherent in this technique. If the gutta percha sections have been well contoured, the apical section is likely to act as a piston and drive any excessive sealer into the periapical tissues. Accordingly, there are modifications of this basic technique that are used with success. Instead of coating the walls of the canal with sealer in advance of insertion of the gutta percha sections, the canal may be left uncoated and each section covered with sealer just

before it is carried into the canal. When the sealer is painted onto the primary section of gutta percha, the blunt end of the apical tip is left uncoated and the gutta percha and cement are seated simultaneously, eliminating the possibility of periapical displacement of cement.

Another modification of the technique may be introduced after the apical section of gutta percha has been firmly and correctly seated. The remaining portion of the canal may be filled by placing into the canal a gutta percha cone that has been rolled in sealer. The cone and the sealer are pressed laterally against the canal wall with a gutta percha spreader and then another cone is placed into the canal. This is repeated until the canal is completely obliterated.

This basic procedure for obturation may be modified in a slightly different manner. The root canal sealer can be omitted. Instead of coating the gutta percha sections with root canal sealer, the sections are dipped into eucalyptus oil. This liquid softens the gutta percha surface and increases its peripheral adaptability. The sections are then inserted into the canal and manipulated in the same manner as described in the preceding paragraph.[3]

Single Cone Method. In this method of obturation, first determine the length of the root. Having completed the necessary calculations, note the width and taper of the canal from the roentgenograpic examination. With these data at hand, select a preformed gutta percha cone which is closest to the established dimensions. If a cone with the necessary dimensions is not available, one may be rolled or fabricated to the correct size. Take a roentgenogram to evaluate the vertical and horizontal fit of the cone. Adjust and pre-fit the cone until it occupies the desired position and fills the space in the canal as thoroughly as possible. At this point, cut the base of the cone level with the incisal or occlusal surface, or make a mark at this level with a sharp instrument. The author prefers the latter method of noting the distance to which the cone should be inserted, since it provides a longer working handle. Then mix the root canal sealer to a thick, smooth and even consistency. The method of applying the sealer to the canal becomes a matter of choice. A fine root canal plugger may be utilized to apply the sealer to the walls of the canal and then the cone is rolled in this sealer and carried to place.

An alternative method is to cover the cone with sealer rather generously to within 4 or 5 mm. of the incisal base, excluding coverage of the blunt end at the apex. The point is then placed into the canal and against one of the walls, and, with an ever so slight vibrating motion, the cone is slowly moved into the canal until the marking on the cone is parallel to the incisal edge or occlusal surface. By slow movement and by holding the point against one aspect of the wall, the operator

will eliminate the possibility of trapping air in the canal. The slight vibrating motion will adapt the sealer to the canal walls and this adaptation is consummated with the thorough seating of the cone.

Make a roentgenogram and study the position of the cone. If the cone position is correct, cut it with a warm plastic instrument at a level comparable to the cervical line and clean the chamber. Should the apex of the cone be slightly short of the desired apical level, cut the cone at the floor of the pulp chamber and apply slight force to the cone parallel to the long axis of the canal. After movement, recheck the position of the cone roentgenographically. The procedure may be repeated and rechecked until the cone is properly seated. If the roentgenogram taken after cementation reveals that the cone projects beyond the apical foramen, the cone should be carefully removed, trimmed and recemented. In most instances, the pulp canal sealers or cements set slowly enough to provide the time required for the additional manipulation.

Once the obturation has been completed, trim the gutta percha and the sealer at the level of the cervical line. This can be done at the same appointment as that used for placing the obturator. In some instances, this can be accomplished with greater ease at a subsequent visit.

Lateral Condensation Method.[2,3] This method is particularly useful when obturating a wide, ovoid, elliptical or ribbon-shaped canal. After determining the length of the root and noting the shape of the canal, select a primary gutta percha point. Place the cone, examine it roentgenographically, and then adjust it until its apex is at the desired level and the body of the cone conforms closely to the outline of the canal. Cut the base of the cone at the incisal or occlusal level or mark it with a sharp instrument at this point. Then remove the cone from the canal and place it in an antiseptic solution. Mix the root canal sealer to a smooth, even, but firm consistency, and coat the walls of the canal with the sealer. This can be done with a fine plugger, file, broach, pick, or even a paper point. Once the walls of the canal have been thoroughly painted with sealer, remove the cone from the antiseptic solution, dip it in alcohol and air-dry it, and then roll it in the sealer. Then work the covered cone slowly into the canal, taking care to hold the cone against one wall. This allows for the egress of air and prevents forcing cement into the periapical tissues.

Once the initial cone is seated, take a roentgenogram to confirm its position (or defer this until after the lateral condensation has been completed). Then carefully place a small spreader into the canal. The object is to wedge the primary gutta percha cone against a wall of the canal and yet not move or dislodge the apical end of the point.

Remove the spreader and insert a small gutta percha cone into the space left by the spreader, taking care to use a cone that is smaller than the spreader. Repeat the procedure until you are no longer able to force the spreader into the canal. Remove the excess filling materials as previously described, and examine the filled canal roentgenographically.

In a modification of the lateral condensation method,[6] the step of coating the walls of the canal with sealer before inserting the initial cone is omitted. No pumping motion is used to carry the point to its seat in the canal. The omission of these aspects of the procedure markedly reduces the possibility of carrying sealer into the periapical area. The author believes that there is merit in this modification. The hazard of overfilling is substantially reduced without affecting the quality of the obturation.

Inverted Cone Method. One of the problems encountered in endodontics results from the necessity to treat teeth with incompletely formed roots. In such teeth, the apical divergence of the walls of the canal makes the placement of a good obturator a complex technical problem.

When such a problem is encountered, instrument the canal until as much of the cervical constriction is removed as possible. This step tends to make the walls of the canal parallel. The more nearly parallel the walls can be made, the easier it will be to place and condense the obturator.

Upon completion of instrumentation, examine the tooth roentgenographically, recalculate its length and note the contour of the canal. Then fashion a gutta percha cone to the size of the canal. Place the base of the cone into the canal first and insert it to the previously calculated distance. Fashion the cone and recontour it until the base is so wide that it will, with slight resistance, pass by the narrowest diameter of the canal. When the cone is correctly placed, either trim it or mark it parallel to the occlusal or incisal surface.

Once the proper relationship of cone to canal is achieved, obturation can begin. Place the cone in an antiseptic solution until the root canal sealer is mixed. After spatulating the sealer, remove the cone from the antiseptic solution, dip it in alcohol, allow it to air-dry, and then coat it with the sealer. Place the cone into the canal, base first, until the tip or the marking on the cone is once again parallel to the occlusal or incisal surface. Place a spreader into the canal and move it so as to adapt the cone against one aspect of the wall of the canal. Upon removal of the spreader, insert a cone, previously trimmed to the same length as the first cone, apex first in the path left by the spreader. Reinsert the spreader, wedge the gutta percha against the

wall and then again remove the spreader. Insert a third cone, just slightly smaller than the spreader, and repeat this procedure until the spreader can no longer be introduced into the canal.

If the canal has been well obturated and the obturator firmly condensed, nothing more is required than removing the coronal portion of the filling. When there is some question as to the thoroughness of the apical seal, an apicoectomy can be performed immediately. This enables the operator to reduce the sharp edges of the root, to trim and further adapt the obturator to the apical orifice, and to remove the residual follicular tissue found in the periapical area.

Silver Cone Method

Jasper's Silver Cone Technique.[4] The introduction of a standardized silver cone—one made to the proportions of a standard canal-enlarging instrument—was a great boon to the practice of endodontics. This silver cone enables the general practitioner to become an "occasional endodontist" and to insert a good obturator into a root canal with relative ease. The silver cone, as suggested by Jasper, makes the selection of a primary cone for obturation a more simple procedure. The silver cone can be removed from the canal, adjusted and reinserted as many times as deemed advisable without fear of breaking or distorting. It facilitates the filling of long fine root canals. Whenever inaccuracies occur after cementation and before the cement sets, the silver cone may be removed, adjusted and then recemented.[5]

The instrumentation of the canal and the therapeutic procedures employed in this technique are essentially the same as for any other aseptic procedure. However, too much emphasis cannot be placed on the necessity for filing away all irregularities from the walls of the canal and producing a canal that is as nearly conical as possible. When the canal being instrumented is thin and thread-like—for example, buccal root canals of maxillary molars, mesial canals in mandibular molars and occasionally a canal in a mandibular incisor[1]—the primary cone may be well adapted to the entire length of the canal (Figs. 5 and 6). Where the walls of the canal diverge in a cervical direction, the canal can be instrumented until the apical third of the silver cone will be well adapted to the pulpal wall. Once the filing of the canal has been completed and during the appointments designated for culturing, the silver point may be tried, roentgenographed and adjusted. Thus when the canal is ready for obturation, the silver cone will be completely adjusted and prepared for cementation.

The silver point can best be sterilized by placing it in a germicidal solution for the recommended period of time. Using the flame
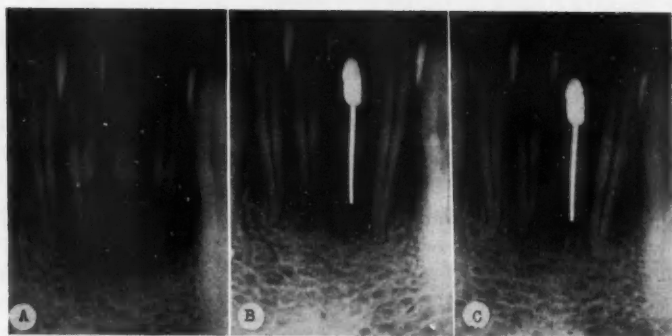
Fig. 5. Roentgenograms made of a mandibular left central incisor obturated with the single silver cone technique. *A*, At initiation of therapy. *B*, At completion of obturation. *C*, One year after completion of therapy, demonstrating complete resolution of apical pathosis.
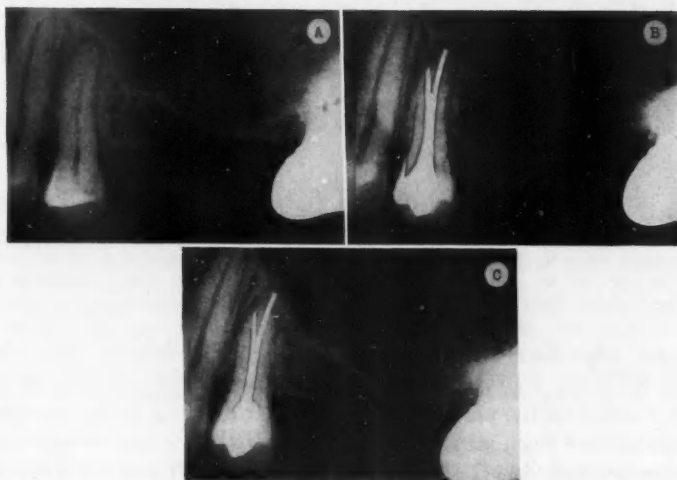
Fig. 6. Roentgenograms of a maxillary left second bicuspid demonstrating obturation of a bi-root bicuspid using the single silver cone technique. *A*, At initiation of therapy. *B*, At completion of obturation. *C*, One year after completion of therapy, demonstrating complete resolution of apical pathosis.

for sterilization has several disadvantages: the heat may melt the tip of the point; it may affect the temper of the metal; and, if the point is not cooled adequately before the sealer is applied to its surface, the heat will affect the setting time of the sealer.

While the silver cone is in the sterilizing solution, mix the root canal sealer and spatulate it to the desired consistency. Then remove the

cone from the solution and dip it in alcohol, allow it to air-dry, coat it with the sealer and carry it into the canal to the previously determined position. If the cone is well adapted to the entire length of the canal, no further manipulation is necessary. When it only completes the seal in the apical third of the canal, obturate the remaining space with gutta percha points as in the lateral condensation of gutta percha technique.

Upon completion of obturation, examine the tooth roentgenographically and note the position of the obturator. Make any minor adjustments that are necessary and then reexamine the tooth roentgenographically. When the obturator is satisfactory, fill the unfilled portion
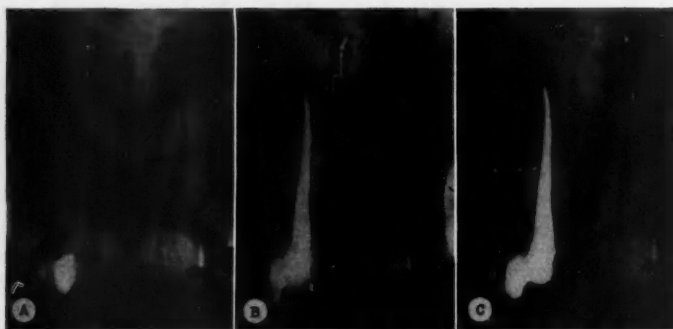
Fig. 7. Series of roentgenograms of a maxillary right central incisor that was obturated using the primary and secondary silver point technique. *A*, At initiation of therapy. *B*, At completion of obturation. *C*, One year after completion of therapy, demonstrating complete resolution of apical pathosis.

of the pulp chamber with oxyphosphate of zinc cement. After the pulp canal sealer and the cement have set, cut off the projecting silver point flush with the periphery of the tooth or reduce it in the chamber to the desired level; usually the cone is cut back to a level comparable to the cervical line. Then refill the chamber with a white cement, which will improve the appearance of the overlying tooth structure.

Primary and Secondary Silver Point Technique for Completely Formed Roots. This method of root canal obturation is particularly adaptable to cases where the canals are large, elliptical or ribbon-shaped. It provides a most thorough procedure for securing a good apical seal and a complete lateral seal throughout the length of the canal (Figs. 7 and 8).

After completing the instrumentation of the canal, begin the process of selecting and adjusting the primary silver point. The adjustment of the primary cone is identical to that described in the Jasper tech-

nique. Once it has been adapted, remove the silver cone from the canal and straighten it. Then sharpen the tip ends of several No. 2 silver points to needle sharpness with a fine sandpaper disk. By adjusting at the base with a sharp scissors, make all the No. 2 points exactly the same length as the primary point. At the appointment for obturation, place the primary and secondary silver points into an antiseptic solution, make the tooth ready, and mix the pulp canal sealer. After
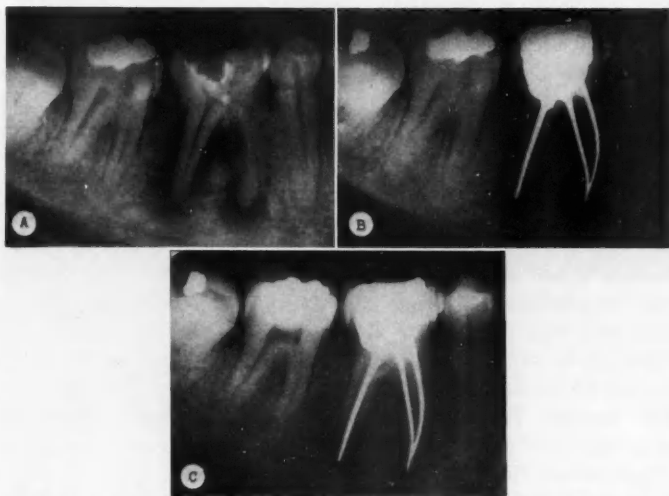
Fig. 8. Series of roentgenograms of a mandibular right first molar. This tooth was obturated using the single silver point technique for the root canals in the mesial root and the primary and secondary silver point technique for the canal in the distal root. *A*, At initiation of therapy. *B*, At completion of obturation. *C*, One year after completion of therapy, demonstrating complete resolution of apical pathosis.

the sealer is ready, remove the points from the solution, rinse them in alcohol and let them air-dry. Coat the primary point with the sealer, except for the flat portion at the apical end, and then insert it into the root canal to the correct position. Then coat a secondary point and insert it into the canal. The secondary point may be inserted until its base is just flush with the base of the primary point. Care must be exercised to prevent the secondary point from going beyond the primary point, as occasionally will happen in a canal that has a ribbon-shaped or elliptical apical orifice. After placing the first No. 2 silver point, coat a second and seat it in the canal. Repeat this process until sharpened secondary points (No. 2 silver points) can no longer be inserted into the root canal.

Using silver points to complete the lateral filling has many advantages over the use of gutta percha. The obturator has a uniform degree of radiopacity. With gutta percha, the actual condensation is achieved with a spreader or gutta percha plugger. The finest and smallest of these instruments cannot approach the size of a sharpened No. 2 silver point and thus does not reach the narrow portions of the canal as does the secondary silver point. Since the prepared secondary points will yield and yet are not compressible, they can be placed in any part of the canal and simultaneously, with hydraulic pressure, drive the sealer laterally with considerable force. Thus the sealer is wedged into the many microscopic interstices of the canal and is thoroughly sealed laterally. The occupation of so much of the space in the canal by silver will minimize any dimensional change occurring during the setting of the root canal sealer. This is an extremely important point, for in the final analysis, no matter how well adapted the silver point or points are to the canal, it is not the silver but the sealer that accomplishes the hermetic seal.

After the obturation has been completed, the tooth is examined roentgenographically, and if adjustments are indicated, they can be made at this time. When the obturator is in correct position, the portion of the silver points projecting into the pulp chamber and beyond can be handled as in the Jasper technique.

The Inverted Primary Point and Secondary Silver Point Method. Just as the canal of the incompletely formed root necessitates the use of the inverted gutta percha cone technique, it also indicates a variation in the silver point technique. After instrumentation of the canal, file the walls until they are as nearly parallel as possible. Select a silver point which, when inserted base first into the canal, will just pass the narrowest diameter of the canal. After calculating the over-all length of the tooth, mark the silver point and insert it in the root canal, base first. When the silver point is in the desired position, examine the tooth roentgenographically and make adjustments in the relationship of point to tooth. When the desired relationship has been obtained, mark the point with a sharp scissors parallel to the incisal edge. Then remove and straighten the cone and prepare an adequate number of secondary silver points as in the preceding technique.

At the appointment provided for obturation, place the primary and secondary points in a sterilizing solution and prepare the tooth for insertion of the obturator. After spatulation of the root canal sealer, place the cones in alcohol and then remove them to dry. Carefully coat the primary point with sealer, taking care not to cover the blunt end of the base of the point. Then insert the point into the canal, base first, to the previously determined position. Coat a secondary point

with sealer and place it in the canal until the incisal extent of the secondary point is just flush with the incisal extent of the primary point. Repeat this procedure until secondary points can no longer be introduced into the canal. It is the circumference of the canal at the narrowest diameter, which in these cases is located at the cervical third of the root, that determines the number of secondary points that
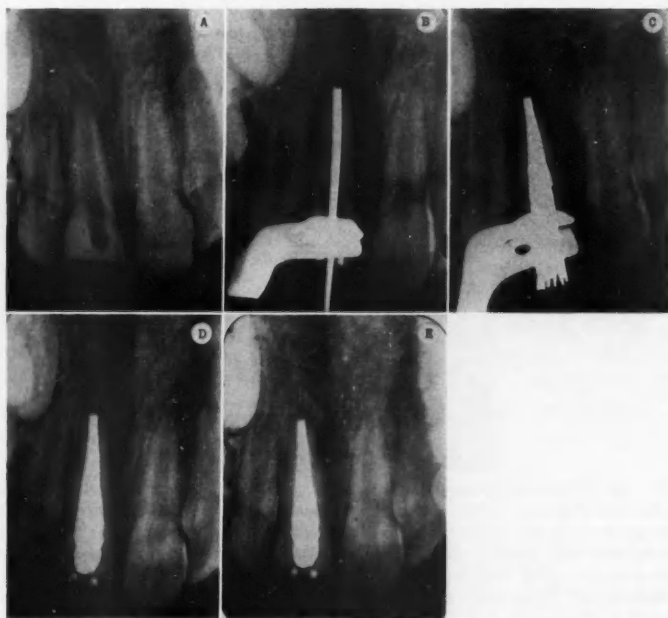
Fig. 9. Series of roentgenograms of a maxillary right central incisor that was obturated using the inverted primary point and secondary silver point method. *A*, At initiation of therapy. *B*, Adjustment of the inverted primary point. *C*, Primary and secondary points sealed in place. *D*, At completion of therapy. *E*, One year after completion of therapy, demonstrating complete resolution of apical pathosis.

can be placed in the canal. Where desired, the number of secondary points that can be placed into the canal can be increased by disking the walls of the primary silver point so that they converge more as they progress toward the apex of the point. This reduces the diameter of the primary point at the level of the narrowest diameter of the canal and thus creates more space for secondary points. This is an important factor if the walls of the root canal diverge markedly in an apical direction. Once the canal is completely obturated, the position of the obturator is checked roentgenographically. When the cor-

rect positioning is obtained, the portions of the silver cones pro-
jecting coronally are reduced in the manner previously described
(Figs. 9 and 10).

It is occasionally suggested or implied that an apicoectomy should
be performed on these teeth at the same appointment as that used
for the insertion of the obturator. The author feels that, if the canal
is well filled and all possibilities of seepage into the canal have been
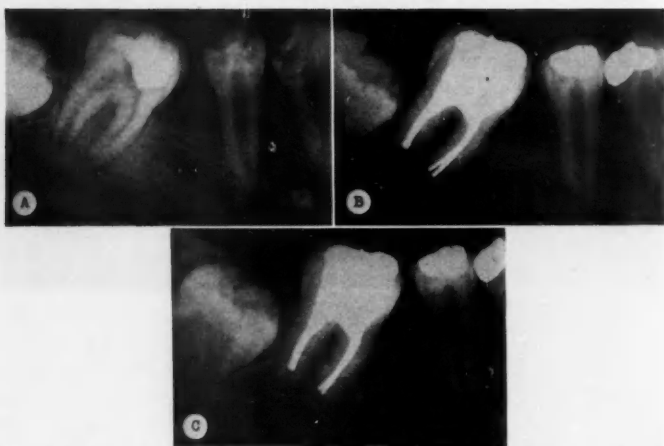
Fig. 10. Series of roentgenograms of a mandibular right second molar. The
mesial roots of this tooth were completely formed and the mesial canals were
obturated using the single silver point technique. The distal root is incompletely
formed and this canal was obturated using the inverted primary point and sec-
ondary silver point technique. A, At initiation of therapy. B, At completion of
obturation. C, One year after completion of therapy, demonstrating complete
resolution of apical pathosis.

eliminated, an apicoectomy at this time is unnecessary and unwar-
ranted. Routine follow-up examinations will reveal that only a small
percentage of these teeth will need surgery. The surgical procedure
can be introduced when roentgenographic evidence demonstrates the
failure of resolution of the periapical lesion.

It is the author's opinion that this technique is markedly superior
to the inverted gutta percha cone method. When using silver points,
there is never the possibility of breaking off a portion of the filling and
displacing it apically. The operator always has positive control of the
position of the obturator until the sealer sets. This provides a reason-
able amount of working time. The secondary points will not change
their shape, only yield in their long axis. They are incompressible and
this provides a method for applying considerable lateral hydraulic

force so that the sealer will not only be carried into the space between all the silver points but also into the irregularities of the root canal.

ROOT CANAL SEALERS

A root canal sealer is a material that is used in a plastic state in conjunction with gutta percha, silver cones, or any other filler or combination of fillers to complete the hermetic seal of the root canal.

Since a filling material is rarely used without a sealer, and a sealer is not used without a filling material or materials, equal care should be taken in selecting both components. In general, the same requirements that have been previously suggested for a filling material are applicable to the sealer. As has been stated in the preceding section, it is the sealer that accomplishes the hermetic seal no matter how well the filling material is adapted to the walls of the canal. Thus in addition to the requirements that have already been enumerated, the sealer must flow readily between all parts of the filler, it must displace air, it should be thin and plastic enough to be forced into all the irregularities, interstices and accessory canaliculi of the pulp canal, it should set slowly enough to provide adequate working time and there should be no dimensional changes upon setting. Once having set, the sealer and filler should be as one, thus effecting a thorough and complete hermetic seal.

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Root Resection and Apical Curettage

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ROOT RESECTION

The term, root resection, implies the cutting off of some portion of a root end for the purpose of removing what is presumed to be pathologic tissue associated with the apex. This operation was first done over a century ago by Desirabodé.

Hermetic Sealing of Canal Prior to Root Resection

In histologic studies of the root ends of resected teeth, Herbert found that such inflammatory reaction as remained in connection with these teeth is strictly limited to the tissues immediately adjacent to the apical end of the root canal filling, and is not found in association with the cut surface of the dentin, which shows evidence of repair over most of its surface. This suggests that so long as the porous apical third of the root is removed, the risk of future infection after a root resection lies much more in leakage from the root canal than in retained infected dentin. If this is so, the success of the operation depends largely on obtaining an adequate seal with an impervious material for the apical end of the root canal.

The case shown in Figure 1 will serve as an excellent illustration to demonstrate that root resection in itself is of no value whatsoever in establishing normal periapical tissues, unless the causative factors responsible for the pathosis have been definitely and permanently eliminated.

Armamentarium for Root Resection

Figure 2 shows the minimal requirements necessary to perform a root resection.

Fig. 1. Failure of root resection due to leakage. A, Periapical involvement 11 months after filling of canal. B, Incomplete bone repair 1½ years following resection; note the leakage between the two gutta percha points "suspended" in the canal.
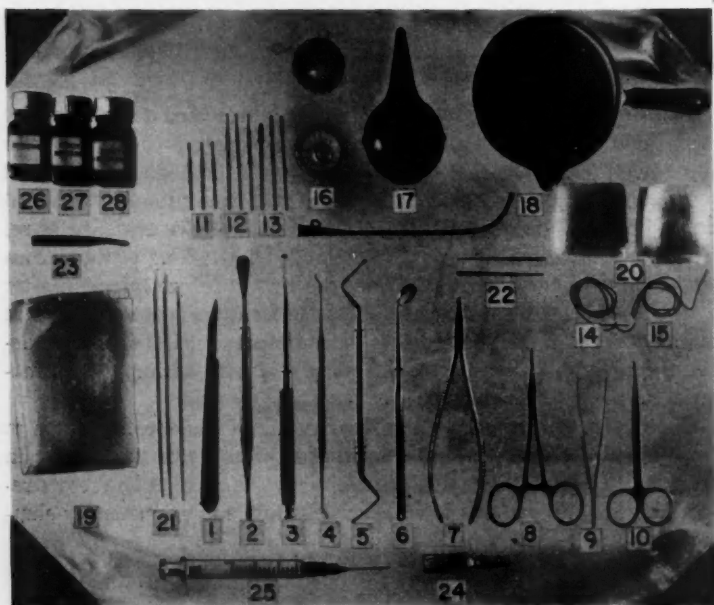
Fig. 2. Armamentarium for root resection. 1, Bard-Parker lance; 2, Hu-Friedy subperiosteal elevator; 3, straight chisel; 4, cow horn explorer; 5, double-ended spoon excavator; 6, small mouth mirror; 7, needle holder; 8, straight hemostat; 9, mouse beak tissue forceps; 10, small straight sharp scissors; 11, four or five No. 560 cross-cut fissure burs for straight handpiece; 12, two Henahen bone drills; 13, antrum burs; 14, cleft palate curved needles; 15, No. 40 cotton thread for suture; 16, two Dappen dishes; 17, ear abscess syringe; 18, porcelain crucible; 19, sterile napkins; 20, sterile sponges; 21, cotton applicators; 22, toothpicks; 23, Howe's silver nitrate; 24, anesthetic solution; 25, syringe; 26, eugenol; 27, tincture of benzoin; 28, tincture of Mercresin.

Technique of Root Resection

After complete anesthesia has been established, proceed as follows for an upper lateral incisor.

1. Start the incision from the middle third of the cuspid. Apply firm and steady pressure on the knife in order to obtain a clean-cut incision through the mucosa and the periosteum. Extend the incision to the frenum, being careful not to cut through it (Fig. 3D).
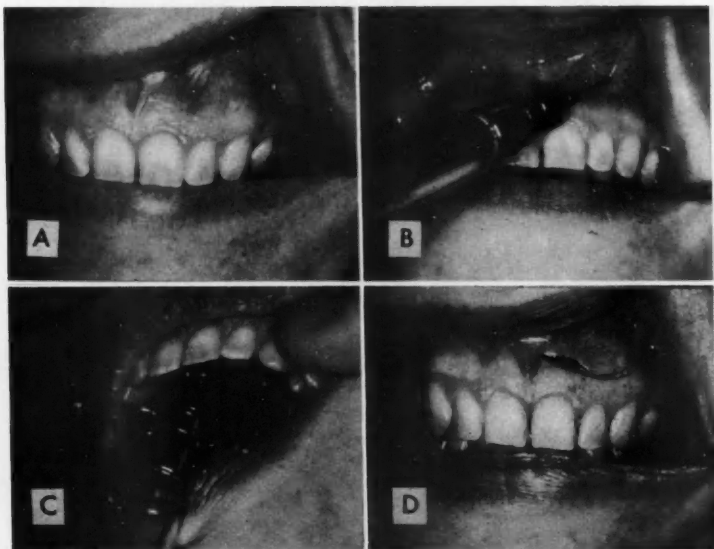
Fig. 3. Technique of anesthesia injection for root resection. A, Frenum attachment is quite low. B, Direction of needle for infraorbital injection. C, Site of nasopalatine injection. D, Extent of incision for an upper lateral incisor.

2. With the aid of the large Hu-Friedy subperiosteal elevator, the mucosa and the periosteum may be elevated simultaneously until a clear view is obtained of the periapical area around the tooth in question.

Appearance of Periapical Region with Resorption of Labial Plate

In the great majority of cases, the labial plate immediately over the periapical area will be found to be partially or completely resorbed, exposing the root tip and the periapical tissues. In these instances, the operator may find it quite easy to enucleate the soft tissue already exposed, with the aid of a double-ended spoon curet, without the necessity of cutting through the labial plate.

In cross-sections made of cadaver jaws to show the relation of the roots of the teeth to the buccal and lingual plates of bone (Figs. 4 and 5), the roots of the maxillary anterior teeth are seen to be in close proximity to the labial plate, whereas considerable alveolar bone may be found between the root and the palatal plate of bone. It is difficult

Fig. 4. Cross sections of upper jaw showing relation of roots to labial plate of bone.

to understand why a periapical lesion will resorb through a cortical layer of bone, such as the buccal plate, rather than follow the path of lesser resistance, namely the spongy bone toward the palatal plate.

Nevertheless, the resorption is usually toward the buccal plate. From the standpoint of bone repair following root resection, it is fortunate that this is so because, as will be stated later, involvement of

Fig. 5. Cross sections of lower jaw showing relation of roots to labial and lingual plates of bone.

the palatal plate of bone is usually associated with incomplete bone repair, resulting in what has come to be known as an operative defect.

Precautions to Be Observed When Labial Plate of Bone Is Intact

1. A cow horn explorer may be used to detect any soft spots in the labial plate, indicating the location of the periapical lesion.

2. If the explorer fails to detect a possible opening, proceed as follows:

    a. Study the roentgenogram of the lesion to be removed.
    b. Note the inclination of the roots of the teeth.

c. Endeavor to determine the length of the teeth.

d. Endeavor to determine the proximity of the teeth to be resected to the normal vital adjacent teeth.

e. If the root of the tooth to be resected is in close proximity to an adjacent vital tooth, extreme care and caution must be observed.

If the opening through the labial plate is inadvertently made to the mesial, or distal, of the actual lesion, there is a good possibility that further exploration could result in the involvement of a normal vital tooth, rather than confining the operation to the tooth originally involved. If the opening through the labial plate is made below the apex of the tooth, it becomes increasingly difficult to distinguish the tooth structure from the surrounding bone. In this event it is quite possible to resect the root at too low a level, and accidentally leave the root tip in position.

Resection of Root Tip

After access to the periapical tissues is obtained, the root tip may be cut off with a No. 560 cross-cut fissure bur. Some operators may prefer to sever the root tip with a sharp bone chisel. It is relatively unimportant which instrument is used. In the writer's opinion and experience, the cross-cut fisure bur causes less trauma, is less likely to split the root, and more likely to produce a clean-cut, smooth root surface (Fig. 6B).

CAUTION: It is very important that the operator be satisfied that the root tip is in clear view. There should be no guesswork at this point. In many instances, the landmarks may become confused through excessive bleeding. Unless the operator is sure of the location of the root tip, the following may occur:

1. He may cut across periapical bone beyond the root tip.

2. He may cut across the root at a level far below the point desired.

3. He may cut into the root of an adjacent vital tooth and inadvertently devitalize a sound tooth.

4. He may not cut the root tip off completely, leaving a sharp spicule of root that will give rise to irritation of the surrounding bone.

Curettage of Periapical Soft Tissue

The removal of the soft tissue with a double-ended spoon in a well circumscribed lesion is a simple procedure. However, in the more extensive involvements, certain complications may arise:
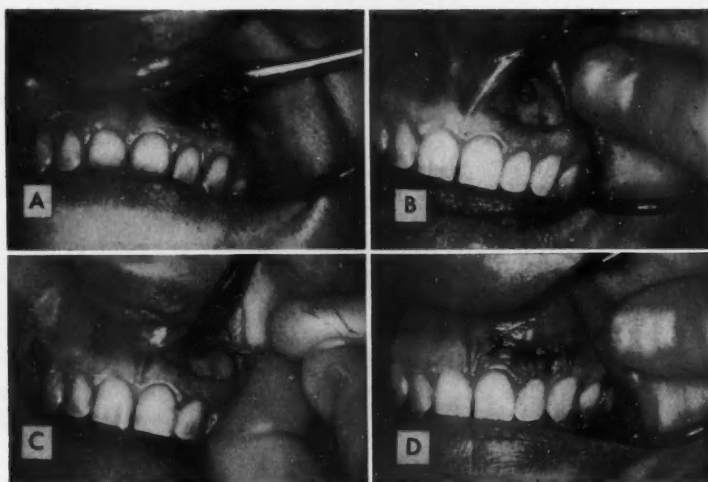
Fig. 6. Root resection. A, Mucoperiosteum is retracted with a Hu-Friedy subperiosteal elevator. B, After curettage and resection, a clear view should be had of the entire area. C, Silver nitrate is reduced over the resected root, using a plain toothpick dipped in eugenol. D, Incision is sutured with cotton thread No. 40.

1. The pathologic tissue may be intimately attached to dense fibrous connective tissue formed as the result of a long-standing fistulous tract. The double-ended spoon curet will not effectively separate the dense fibrous connective tissue from the soft tissue. The scalpel may be more useful in this instance.

2. The abnormal tissue may extend around the lingual to an adjacent vital tooth.

3. The abnormal tissue may be so extensive as to come into close contact with the floor of the nasal fossa. In such cases, extreme care should be exercised. An accidental perforation into the nasal cavity could result in a nasomucous fistula. In order to avoid such an accident, careful study should be made of the roentgenogram of the region involved and the possible relationship of the periapical involvement to the nasal floor should be considered.

The student or the inexperienced dentist who is doing his first resection becomes so engrossed with the novelty of doing an apicoectomy for the first time that he is quite apt to forget, for the moment, the anatomic relation of the tooth to vital landmarks.

4. The periapical soft tissue may be so extensive as to come into close contact with the nasopalatine canal. As pointed out under 3, the same care must be exercised in order to avoid damage to an important anatomic area.

5. The periapical soft tissue may extend toward the palatal plate of bone. If and when the palatal plate has been perforated during the removal of the soft tissue, complete bone repair will be hindered by the development of an operative defect, which will be discussed under *Apical Curettage.*

Treatment of Tissues During Resection

The degree of postoperative pain and swelling is almost in direct proportion to the trauma to the tissues incurred during curettage. Injudicious or prolonged scraping of bone and inconsiderate treatment of the soft tissues will result in unnecessary pain and swelling. Bone drills should be used as sparingly as possible, and revolved at such speeds as to produce the least amount of heat. The tissues must be treated at all times with as much care as possible in order to avoid or minimize unnecessary discomfort to the patient.

Bleeding. Once the incision has been made through the labial mucosa, bleeding is constant during the course of the operation. The extent and duration of bleeding depend largely upon the individual patient. In some cases one may encounter comparatively little bleeding, whereas in other cases the entire operation may be made increasingly more difficult on account of it. Methods of controlling bleeding are the following:

Gauze Sponges. Perhaps the simplest means of controlling bleeding is the use of gauze sponges. It should be remembered, however, that injudicious use of sponges may cause tissue irritation. If the sponges are constantly pressed against the bleeding parts, the threads in the gauze may act as sources of irritation and contribute to postoperative pain. If the gauze sponge is twisted into the form of a spiral and gently pressed into the bleeding socket with the end of the curet, the sponging operation will succeed more rapidly.

Aspirators. Various types of suction devices are on the market for the purpose of aspirating blood from the field of operation. These include saliva ejectors on the dental unit, suction hoses attached to a water faucet, and power-driven suction pumps.

Irrespective of the type of aspirator employed, its effectiveness is largely dependent upon the skill of the assistant using it. In some cases, it is difficult for two people to gain access to the area at the same time. Then again, there may be intervals from the time the aspirator is withdrawn and the time the operator resumes the curettage, so that sufficient blood will reenter the area to obstruct the field of vision. Nevertheless, the aspirator is preferred to the gauze sponge in most instances.

Resection of Fractured Roots

In the great majority of fractured roots, the pulps will remain vital indefinitely. Cementum and bone will unite the fractured portions of the root, and, to all intents and purposes, the tooth will function normally. However, occasionally the blow causing the fracture may result in pulp necrosis. If the fracture occurs halfway down the side of the
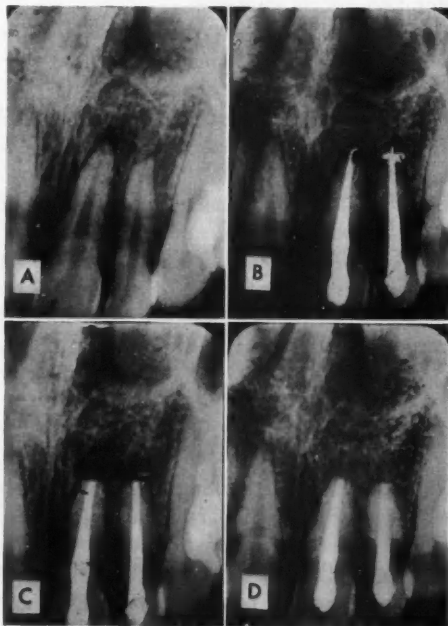
Fig. 7. Root resection of fractured tooth. A, Pulp death in upper right central with chronic alveolar periodontitis, the upper right lateral showing fracture of root on apical third. B, Canals were purposely overfilled because resection was contemplated. C, Teeth immediately after resection. D, Complete bone repair after 8 months.

root, there may be an insufficient amount of tooth structure remaining for support. Extraction is the only alternative. However, if the fracture occurs in the apical third of the root, it can be successfully removed by performing a root resection.

Some Indications for Root Resection

1. Teeth with extreme apical curvatures which cannot be adequately instrumented and hermetically sealed to the dentocemental

junction. These should be resected to the level where the canal is well filled.

2. Teeth in which periapical pathosis is the direct result of irritation from excess filling material. A resection or apical curettage will make it possible to remove the overextended filling.

3. Teeth in which instrumentation has resulted in accidental perforation of the root in the apical third. These must be resected back to where the perforation can be eliminated.

4. Periapically involved teeth in patients who cannot spare the time for conventional endodontic treatment. These can be managed in a one-sitting operation by the use of acids.

5. Teeth in which a root canal instrument has been broken off in the apical third. In most cases, root resection is the only way by which a broken file can be removed.

6. Strategic teeth with poor canal fillings which, in the course of removal, might be forced beyond the apex.

7. Periapically involved teeth which for some reason did not respond to endodontic treatment. Resection in these cases is always a hazardous procedure. The very fact that the endodontic treatment has failed is indicative that the causative factor has not been removed (Fig. 1).

Rate of Bone Repair Following Root Resection

The time required for complete filling of the bone cavity left following resection is roughly proportional to the size of the cavity itself. There are no hard and fast rules to follow, however. In periapical lesions of average size, from 8 to 12 months are required for osteogenesis. Therefore, it becomes hazardous to judge the progress of a root resection unless one can visualize the sequential series of roentgenograms taken at periodic intervals under constant exposure factors.

Procedure for Postoperative Check-up Roentgenograms

1. The original roentgenogram should be used as a guide for comparison with subsequent check-up films.

2. A record of exposure time and angulation should be made of the roentgenogram taken immediately following resection.

3. ALL SUBSEQUENT ROENTGENOGRAMS SHOULD BE TAKEN UNDER IDENTICAL EXPOSURE FACTORS, IN ORDER TO HAVE A BONE BACKGROUND OF CONSTANT DENSITY TO COMPARE WITH THE DENSITY OF THE NEW BONE AS IT IS BEING LAID DOWN IN THE RESECTION CAVITY.

APICAL CURETTAGE

Apical curettage is a term applied to the surgical removal of tissue in the periapical region without resection of the root itself. The root tip may be smoothed off if there is excess filling. Bone repair will take place just as rapidly as when the root itself has been resected.

Operative Defects Following Resection or Apical Curettage. Periapical lesions frequently extend in a palatal direction. During the curettage of the periapical tissue a portion of the softened palatal plate of bone may be inadvertently removed, along with the granulation tissue. Roentgenograms taken immediately after the resection will reveal a dark or nearly black central zone in the region just curetted. After the remainder of the periapical tissue has calcified into bone, a central dark area may still persist at some distance from the resected root ends. This dark area is due to incomplete repair of the palatal plate of bone, and is called an *operative defect.*

Both root resection and apical curettage have been proven to be successful means of removing a periapical lesion and restoring the surrounding tissues to normal. However, these techniques have been advocated in far too many cases in which non-surgical procedures would have been equally satisfactory. The writer has shown[1] that most periapical lesions are nothing more than granulomas, surrounded by granulation tissue. These are tissues of both defense and repair and, if given an opportunity through proper endodontic treatment, bone repair will take place in less time than when these tissues are removed surgically. Careful diagnosis and treatment planning will provide indications and contraindications for both the surgical and the non-surgical approach to the management of teeth with periapical involvement.

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Coronal Restoration of the Treated Pulpless Tooth

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INHERENT CHARACTERISTICS OF THE PULPLESS TOOTH

Conditions unique with the pulpless tooth exist and must be considered during procedures concerned with its coronal restoration following endodontic therapy.

Decreased Moisture Content and Brittleness. Brittleness attributed to decreased moisture content is characteristic of a pulpless tooth. A cursory review of the literature does not reveal reports of any investigations that substantiate the claim that there actually *is* less moisture in the substance of the pulpless tooth, however. It appears quite basic, though, that the moisture content of the dentinal tubuli of the vital pulp tooth has its source in the pulp. With this source eliminated in the pulpless tooth it logically follows that a decrease or even a complete disappearance of the dentinal moisture occurs. Despite lack of scientific evidence of the relationship between decreased moisture content and increased brittleness in the pulpless tooth, it must be readily admitted that the latter condition is a characteristic of the pulpless tooth.

Loss of External and Internal Tooth Substance. It is obvious that the vital pulp of a tooth has its highest degree of protection when its crown is intact. The main causes of deviation in the integrity of the crown—and as a consequence, pulpal injury and disease—are caries and/or trauma. These causes likewise are responsible for a decided loss of external enamel and internal dentin. Necessary instrumentation incident to endodontic therapy results in the removal of even more tooth substance. Therefore, when endodontic procedures are completed, the pulpless tooth has a marked decrease in remaining sound tooth structure. This is obviously accompanied by decreased possibilities for meeting adequate resistance and retentive requirements for the subsequent restoration.

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These inherent characteristics of the pulpless tooth must be considered and compensated for in the procedures, methods, and materials utilized in the restoration of its crown. Failure to do so will result in eventual fracture of the inadequately restored crown. This will reduce it to such a complex condition that its further restoration is impossible. Precautions pertaining to the frequent tendency of the pulpless tooth for fracturing have also been discussed by Coolidge[1] and Grossman.[2]

RESTORATIVE PROCEDURES AND MATERIALS

Research and development in restorative dentistry, assisted by increased knowledge of dental materials, has provided dentistry with the highest effectiveness in the coronal restoration of teeth that it has ever possessed. A consolidation of present-day endodontic efficacy and restorative procedures supported by valid principles of the science of dental materials can only result in the restoring of a decidedly increased number of pulpless teeth to a condition of function in the patient's dentition. A lack of such consolidation of activities or a decrease in the quality of either will contribute to the eventual loss of the tooth or teeth concerned.

This article discusses some of the improved methods of restoration, and their utilization of the recent developments in dental materials, which are most useful in the restoration of the crowns of pulpless teeth subsequent to endodontic treatment.

It has been pointed out[3] that although the gaining of direct access to the root canal of an anterior tooth by way of a lingual opening necessitates only a comparatively minute subsequent restoration, the frequency of use of the lingual opening makes its restoration of great importance.

Coronal discoloration following root canal treatment through a lingual opening need not occur as frequently as it does. The discoloration is often caused by failure to remove all of the coronal pulp tissue, especially that portion contained by the pulp horns and the remainder of the incisal area of the pulp chamber. An awareness of its possible presence in that location must be utilized to overcome the difficulty of its detection. Discoloration can also be caused by failure, following the filling of the canal and before restoring the lingual opening, to remove all of the canal filling material and any other discoloring material from the pulp chamber. If decided discoloration of the crown of the tooth is already present, bleaching procedures are advantageous at this stage. Otherwise, with the pulp chamber thus cleaned, the placing of a cement lining of a shade compatible with the shade

of the tooth, followed by the insertion of a restorative material of equally agreeable shade and with non-discoloring tendencies, will do much to minimize the possibility of later crown discoloration.

Quite often silicate cement or amalgam is used to restore the lingual openings in the anterior teeth. Silicate cement is readily soluble and amalgam tends to discolor; both characteristics contraindicate the use of those materials in the location under consideration.

Self-curing Resins

The self-curing resin gave promise of being an ideal restorative in some types of cavities. However, clinical usage and investigation in the dental materials laboratories demonstrated certain disadvantages in the material. Its low degree of hardness, its dimensional instability, and its low modulus of elasticity were responsible for diminishing the original enthusiasm for its *general* use. In the lingual opening, because there is little or no occlusal stress, hardness and a high modulus of elasticity are not required of the restorative material. The resin's high degree of insolubility and more favorable color characteristics contribute to its advantage over silicate or amalgam in the lingual opening. A further advantage in such use of the material is the fact that its dimensional change can be controlled through the use of a non-pressure neutralizing or contraction-compensating technique which was developed by Nealon.[7]

Amalgam

Dental amalgam is the most frequently used restorative material, and it is excellent when handled properly and with a thorough knowledge of its physical properties. When abused, however, it will lead to irreparable coronal damage.

Healey and Phillips[4] have pointed out that the main factors which contribute to amalgam failures are (1) improper cavity preparation, which was the causative factor in 56 per cent of all of the failures examined by them, and (2) faulty manipulation of the amalgam, or its contamination at the time of insertion, in 40 per cent of all failures.

It was apparent from this study that the clinical success of amalgam restorations is dependent upon many factors, of which 96 per cent could have been controlled by the dentist. Careful cavity preparation, proper manipulation of the amalgam and restoration of normal anatomic contour can reduce clinical failure of amalgam to a minimum.

Among indications for the use of amalgam in the restoration of the pulpless tooth following treatment are (1) marked undercutting from
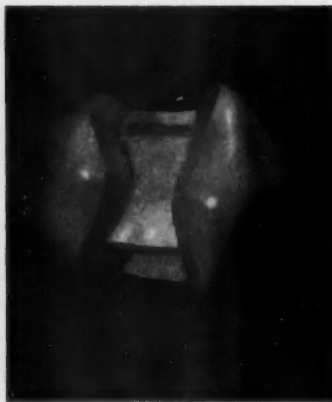
Fig. 1.   Fig. 2.

Fig. 1. Proper MOD amalgam cavity with provisions for adequate bulk of restorative material for strength and for required retentive and resistance forms.

Fig. 2. Finished amalgam restorations including those for two endodontically treated bicuspids.

caries or instrumentation which militates against the use of a gold inlay; (2) economic considerations; and (3) a time factor or decreased patient availability.

On the other hand, amalgam should not be used for this restorative purpose (1) if strengthening or support of previously weakened cusps or lobes is needed, or (2) if discoloration probabilities will interfere with favorable esthetic results.

Gold Inlays

Through the use of gold inlays to restore the lost structure of the crowns of pulpless teeth, it is possible to benefit from continued developments in investment materials, inlay golds, casting techniques, and procedural concepts. Earlier, empiric procedures utilizing materials of inconsistent and inadequate compensatory behavior resulted in castings the fit and use of which were only in accordance with the conscience and integrity of the dentist. In part, the investigation into the behavior of casting materials by the American Dental Association Research Fellowships in the United States Bureau of Standards has resulted in the setting of specifications for casting materials. The adherence to these specifications by the manufacturers and the utilization of improved techniques for their use provide present-day scientific castings of decidedly improved fit.

Cavity Preparation. A model of a cavity preparation for a bicuspid MOD is shown in Figure 3. This illustrates the required features of this type of cavity preparation. Owing to the loss of tooth substance between the cusps or lobes of the tooth through caries and instrumental removal for the purpose of access to the canals, a predisposition for the splitting away of one or the other lobes exists in the treated tooth. This possibility would be further enhanced by the eventual increased brittleness in the pulpless tooth. To counteract both of these conditions, the cusps must be supported and strengthened by reducing them and by subsequently capping them with the gold of
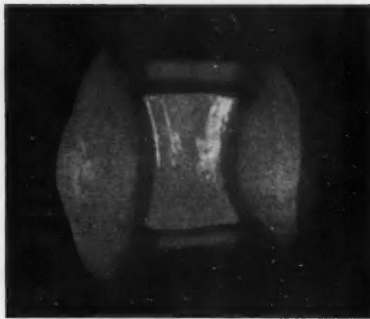
Fig. 3. Model of MOD inlay preparation showing required features including adequate cusp protection, flat cervical seats, and definite cervical bevels.

that portion of the inlay. The exaggerated reversed or inverted bevelling of the cavosurface angle of each cusp provides a gripping together of the lobes by the inlay and thus gives additional resistance to the splitting action of the force of mastication upon the tooth. The provision for dissipation of occlusal stresses through the inlay into the cervical areas requires that definite and flat cervical seats be provided in the preparation. The lost interlobular tooth substance is represented in the model as having been replaced by a cement base shaped to the required axial and pulpal wall forms. The cervical margins are well bevelled. The reproduction of those bevels in the wax pattern gives assurance that the cervical margins, where access is sometimes difficult, have been adequately covered. Although the method of choice in the making of the wax pattern for a single inlay is determined by the individual dentist, it is the author's opinion that the direct method not only saves time but also results in better qualities in the pattern.

Multiple Posterior Inlays. As a recent development in restorative dentistry, multiple inlay or quadrant inlay construction is now almost routinely used. This new procedure utilizes elastic impression ma-

terials—reversible and irreversible hydrocolloids, rubber base materials and silicones.[5,6,8,10]

The inlays shown in Figure 4 include those for the restoration of two previously treated bicuspids (Fig. 5) and were made through the use of a multiple inlay procedure utilizing a reversible hydrocolloid impression material. The illustration of the completed inlays points out the adequate occlusal protection and the provision for support of the previously weakened buccal and lingual lobes.

Fig. 4.   Fig. 5.

Fig. 4. A quadrant of inlays constructed by the multiple procedure and including those for the restoration of two treated bicuspids.

Fig. 5. Roentgenograms of canal fillings of two bicuspids restored by inlays shown in Figure 4.

Multiple Anterior Inlays. Although not as frequently advocated for use in the anterior region, the multiple procedure is also readily applicable there. Figure 6 is a view of carious anterior teeth which were so restored. The pulps of the central incisors were involved by caries and required endodontic treatment. Because of the complex occlusion and the patient's indifference to the appearance of gold, it was felt that these teeth would be more effectively restored through the use of inlays than by the use of perhaps more esthetic materials.

Class IV, Lingual Lock Inlay. When properly made, the type of cavity preparation shown in Figure 10 can be used for the inlay restoration of the tooth with a vital pulp. The definite labial wall, the cervical groove, and the sufficient depth and nearly parallel walls of the lingual lock provide adequate lingual resistance to occlusion and, as a consequence, maximal retention for the inlay. However, owing to the necessity for better access to the root canal when the use of this type inlay is planned for a pulpless tooth, the lingual lock must be modified as shown in diagrammatic form in Figure 11. On the left
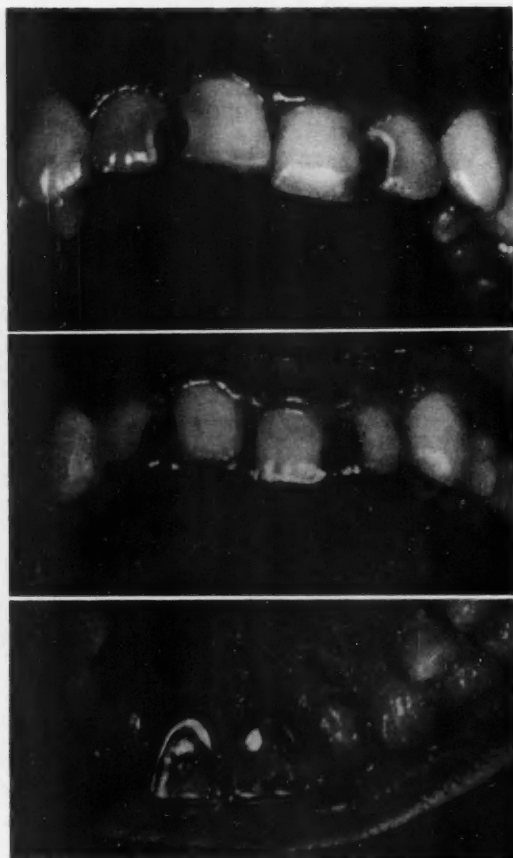
Fig. 6 (*Top*). Preoperative view of extensive carious lesions in anterior teeth.
Fig. 7 (*Center*). Inlay restorations made following endodontic treatment of both central incisors, and utilizing the multiple procedure.
Fig. 8 (*Bottom*). Lingual view of anterior inlays.

is a representation of the lingual aspect of the conventional cavity preparation shown above. The middle portion of the diagram shows a modification of the lingual lock to provide for a better approach to the root canal. Upon completion of obturation of the canal and the removal of the excess gutta percha and root canal sealer from the coronal portion of the tooth, the proper placing of a cement base will permit the withdrawal of the wax pattern in a lingual and slightly incisal direction. On the right is a double-curve labial outline which is advocated for giving a better esthetic result to the completed inlay.
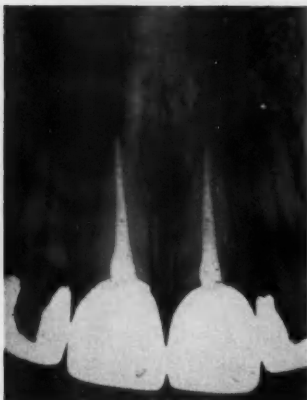
Fig. 9.   Fig. 10.

Fig. 9. Postoperative roentgenogram of multiple anterior inlay case.
Fig. 10. Model of cavity preparation for class IV inlay restoration of a tooth with a vital pulp.

Class IV Lingual Lock Inlay

1. Conventional lingual   2. Enlarged lingual lock   3. Labial Outline
   lock

Fig. 11. Diagrammatic representation of adaptation of conventional class IV cavity preparation for use in restoration of a treated pulpless incisor.

FRACTURED INCISORS

The routine in the office of the general practitioner is often interrupted by the unexpected appearance of a young patient with a fractured incisor or incisors. The amount of lost coronal structure varies, and in cases that require endodontic therapy, the restoration of the crown following treatment requires special consideration. Owing to the extent of the fracture, a jacket crown usually must be used; for the same reason, the retentive possibilities for such a restoration are lessened by the decreased incisogingival length of the traumatized crown. An additional complicating factor is the large pulp chamber,

which is typical in young persons and which adds to the weakening of the remaining portion of the crown when it is further reduced to a jacket crown preparation. To overcome both of those complications a metallic post supporting a gold core can be used. The post of gold-platinum-palladium alloy is fitted into the root canal after enough of the canal filling has been removed to accommodate the length of the eventual jacket crown. The post is permitted to extend out of the canal and into the fractured area sufficiently to permit the waxing up of an incisal portion of the preparation. The casting (Fig. 12) is finished and seated and the jacket preparation completed (Fig. 13).
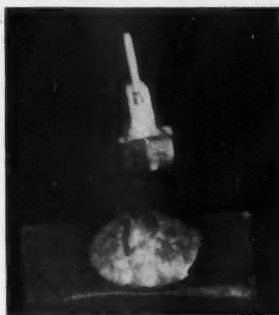
Fig. 12.   Fig. 13.

Fig. 12. Gold core casting and retentive post for augmentation of residual tooth structure.

Fig. 13. Jacket crown preparation with increased strength and retentive features provided for the restoration.

In the illustrations of the casting, it is seen that one surface of the post was purposely left exposed to accommodate the escape of excess cement from the canal at the time of the seating of the post and core. It can also be seen that in the waxing of the core, the wax was carried well up onto the canal portion of the post. This gives additional adherence to the post for the core. The completed preparation shown in Figure 13 is a basic one. In it, both the original retentive deficiencies and the decreased strength in the fractured tooth have been overcome by the post and core.

Figure 14 shows two fractured and pulpally involved teeth which were treated endodontically (Fig. 15) and restored (Fig. 16) in the manner just described. Although posts and cores were not needed to increase the incisogingival length of the jacket preparations for retentive purposes, they were needed to strengthen the teeth which were decidedly weakened by large pulp chambers.
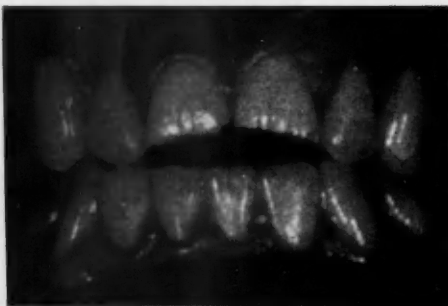
Fig. 14. Fig. 15.
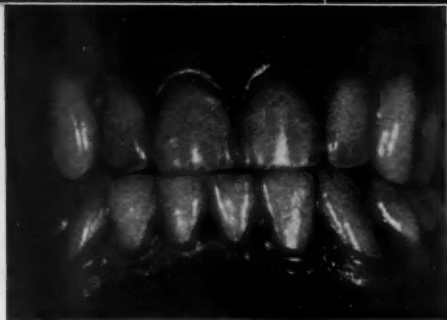
Fig. 16.

Fig. 14. Fractured and pulpally involved incisors.

Fig. 15. Postoperative roentgenograms of endodontic treatment, also demonstrating large size of pulp chambers.

Fig. 16. Jacket crown restorations.

In the construction of jacket crowns, one must consider esthetic requirements, especially shade matching and reproduction of normal anatomic contour. The first of these requires experience and an almost innate sense in color selection. In the latter, a knowledge of normal anatomic tooth contour and an ability to reproduce it are essential. The reproduction of correct labial surface lobular appearance by properly placed developmental grooves provides a normal esthetic appearance in the jacket crowns.

FULL METALLIC COVERAGE

When the crown of the pulpless tooth is so greatly broken down by either caries or trauma and additionally by endodontic instrumenta-

tion that it is impossible to restore it effectively through the use of amalgam or inlays, it is necessary to resort to full metallic coverage of the crown. The difficulty in maintaining a sterile field during treatment of such cases is readily apparent. Sommer et al.[9] have described pre-endodontic methods utilizing various kinds of metallic bands to replace missing walls of teeth and/or to seal off deep cervical portions. Such procedures are necessary to facilitate treatment under aseptic conditions.

The use of a complete gold crown provides maximal protection to the weakened crown of the treated pulpless tooth. Esthetic considerations prohibit the use of the all-gold restoration toward and including the anterior part of the mouth. In those locations the strength of the gold crown can be used and the objectionable appearance can be overcome by veneering the buccal or labial portion of the crown with acrylic resin or porcelain.

SUMMARY

Endodontics at the present time has its highest degree of effectiveness and success in the maintenance of the pulpless tooth in the mouth in a state of health. This is due to the development of new pharmacologic remedies, treatment concepts, procedures, and techniques for the effective sterilization of the root canal and its subsequent obturation. Because of the utilization and application of knowledge of biologic sciences, doubt no longer exists as to the effectiveness of endodontic therapy or as to why and how the treatment outcome occurs.

The return of the pulpless tooth to a state of health must be augmented by a return to function. Fortunately, improved restorative procedures utilizing materials, the validity of which has been proved by scientific methods in the dental materials laboratories, enable us to complete the rehabilitation of the pulpless tooth.

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The Bleaching of Discolored Teeth

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Discolored teeth are usually non-vital. The vital pulp may be said to serve a cosmetic function in the maintenance of the proper hue, shade, and translucency of teeth. However, vital teeth may also be discolored owing to such causes as hypoplasia, opalescent dentin (dentinogenesis imperfecta) and mottled enamel (dental fluorosis). Eliminating the genetic and environmental factors, in endodontics we are concerned with only two causes of discoloration: (1) exogenous pigmentation and (2) endogenous pigmentation.

Exogenous Discoloration

Certain food pigments, smoking and tobacco chewing may discolor teeth. In these conditions, the frequent use of an effective dentifrice and frequent thorough prophylaxes are our only practical weapons. The patient should avoid the staining substances if possible.

Endogenous Discoloration

This may occur under various conditions:

1. Teeth may gradually change in color with age, owing to deposition of secondary (physiologic) dentin or adventitious dentin following the placing of deep restorations.

2. The coronal pulp may calcify completely after mild trauma or restorative stimuli. Should this occur in those under 30, the change is evident because the adjacent teeth are relatively light in color.

3. When icterus accompanies a disease in the first 5 years of life, bilirubin may discolor the developing teeth. Icterus neonatorum affects the deciduous teeth.

4. Severe enamel fluorosis is associated with a characteristic brown stain, which may be at least partly exogenous in origin.

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5. As noted above, pulp degeneration is the primary offender. The degree of discoloration is most pronounced in young patients.

The total mechanism and important contingent factors must be understood before we can predict any success by whatever bleaching method we may wish to employ. Even though a tooth may be effectively bleached, it may discolor again (see Table 1).

Clinically, and by microfiltration through dentin sections, the author has been able to observe and demonstrate the passage of the hemoglobin of the blood to the amelodentinal junction. The so-called "pink

TABLE 1. *Bleaching Effectiveness to Be Expected*

IF DISCOLORED BEFORE	PERCENTAGE DISCOLORED AFTER SUCCESSFUL BLEACHING*	PROPOSED TREATMENT
Age 10	Over 90%	Plastic jacket crown
Age 20	About 75%	Bleach (then porcelain crown if necessary)
Age 30	About 50%	Bleach
Age 40	About 25%	Bleach
Age 50	About 5%	Bleach (intensity of "fallback" barely noticeable)

* "Fallback" may vary from very slight to complete. In order to arrive at the foregoing, the author relies on an endodontic shade guide. Before bleaching, he records the general shade (middle third) of the discolored tooth and that of the adjacent teeth. The S. S. White color matching guide is most suitable for this purpose. The discoloration is most frequently found to be comparable to the E to K series and shades 22, 26, and 27. The "fallback" is related to intensity and duration of original discoloration.

tooth," which may follow trauma, is caused by extravasation of the blood, especially the hemoglobin, throughout the dentin. Unattended, and in the absence of ensuing infection, these teeth will rapidly change to dirty gray or gray-black. The author has observed possibly ten cases of "pink tooth," and in every instance the pulp damage was irreversible. All neural tests may remain favorable for nine months but the pulp ultimately succumbs. Hence, it appears that immediate extirpation of the pulp is advisable in all such cases. The "pink tooth" is most difficult to bleach and the prognosis is unfavorable if it is allowed to become very dark. This condition is peculiar to the 8 to 20 year old age group. (Compare this age range with the lasting effectiveness of the bleach as previously given in Table 1.)

The Pigments of Discoloration. The various protein degradation products, particularly the ptomains and hematoporphyrins, assume some part in the mechanism of discoloration. However, it appears that the formation of iron sulfides (from cysteine, cystine, and iron derived from the hemoglobin of the blood) and other sulfide conjugates are

principally responsible for severely discolored teeth. Leaking margins of the coronal restoration which permit proteins from the external environment to invade the dentin may be responsible for discoloration following root canal obturation. Metallic stains in the dentin from coronal restorations or from some obsolete drugs used in the medication and filling of the root canal are most difficult to bleach.

Anatomic Aspects of Discoloration

The larger the pulp the greater the intensity of the discoloration, owing to the greater volume of degenerated pulp substance and its greater proximity to the exterior of the tooth. Endogenous pigmentation is not necessarily uniform. Often the cervical, middle, and incisal thirds of the crown differ in intensity or character of discoloration.

Histophysical Variations. A direct relation has been found between the age of a tooth and its resistance to bleaching. As would be expected on the basis of the foregoing, teeth in young patients are readily bleached. Unfortunately, teeth that are rapidly bleached (often in a matter of minutes) may revert to almost the original discoloration in a few months. In this respect, the enamel and dentin appear to act like the ceramic filters used for sterile bacteriologic preparations. In actuality, the teeth from different individuals appear to possess various "porosities" or at least act as diffusion strata from the prepared pulp chamber to the external enamel surface.

METHODS OF BLEACHING

There are many techniques for bleaching teeth. Only three methods used by the author and found to be effective will be described. Certain basic concepts should be recognized, viz.:

1. After therapy a conspicuous tooth should be overbleached.

2. The overbleaching is only temporary. Within 3 days to 3 weeks the tooth will regain its normal shade and hue.

3. If an agent such as sodium hypochlorite, applied routinely as a cleansing and disinfecting agent, bleaches the tooth satisfactorily, use of stronger agents is unnecessary.

4. The medication of a tooth may be accompanied by a sustained bleaching agent in order to minimize the number of appointments.

5. Thermocatalytic bleaching (explained below) may be employed should only a single appointment be feasible.

The Hydrogen Peroxide or Hypochlorite Bleach

It is desirable to use hydrogen peroxide or hypochlorite bleach after the obturation of the canal in all teeth which are *not* discolored. This

is done to avoid discoloration after endodontic therapy. The gutta percha, the sealer, and the silver point (if used) must be cut back to a point 2 to 3 mm. apically to the labial gingival line. A small cotton pellet is then dipped in the hydrogen peroxide–ethereal solution and introduced into the canal. Many prefer to use a 30 per cent aqueous solution of hydrogen peroxide (Superoxol) because it lacks the inflammable and explosive character of the ethereal solution. However, the author prefers the ethereal hydrogen peroxide because of its lower surface tension and better penetration. This is allowed to remain for a few minutes. The hydrogen peroxide–ethereal solution is usually used in patients over 30 years of age; under 30, sodium hypochlorite frequently will suffice. The pellet is removed and the cavity is barely dried with a stream of air. The coronal portion of the tooth is then filled with light ivory or white cement, or a combination of the two. Occasionally, the labial facade of enamel is so thin and translucent that a near-perfect match is essential. A rubber dam is always used.

The Sustained Hydrogen Peroxide–Ethereal Bleach

The sustained hydrogen peroxide–ethereal bleach is used, preferentially, incident to treating a tooth and bleaching it simultaneously. The scheme by which such a dual effect may be realized is shown in

Fig. 1. A crown prepared for bleaching; all severely discolored dentin has been removed. A, Sterile paper point moderately saturated with germicide. B, Fluffed and firmly packed sterile cotton pellets saturated with 33 per cent hydrogen peroxide–ethereal.

Figure 1. The lingual approach is closed only with a rubber base temporary stopping. The latter, in the act of mastication, should slightly "telescope" into the lingual approach. If the closure is properly made, the pressure will cause the bleaching agent to diffuse throughout the

dentin *and* enamel. Some may question the use of temporary stopping as a seal for the canal during root canal treatment, but in the author's experience it is possible to prepare the canal for filling without additional sittings. While the bleaching agent has irritating properties, it does not cause tenderness to develop during this treatment. Simple medicaments that do not react with the bleaching agent are employed. Avoid silicone-base antibiotic pastes or creams for most effective bleaching. The bleaching effect is complete in 24 to 36 hours and the chemicals may be removed a day or two or a week or two thereafter if necessary. Flood the chamber with sodium hypochlorite, dry, and fill with a light-colored cement. Always use the rubber dam.
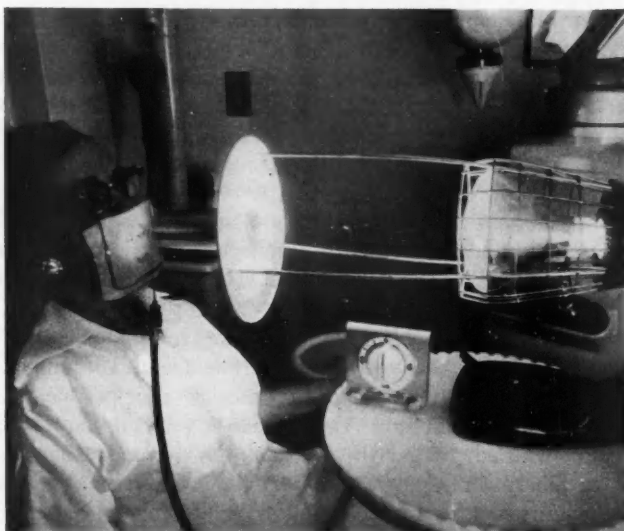
Fig. 2. The general arrangement for the thermocatalytic bleach. The patient is quite comfortable during the procedure. There is no need for Therminon glasses or for placing a towel over the eyes. One minute is all the time required for each centimeter that the speculum is removed from the foremost portion of the clamp. The optimum appears to be 5 minutes at 5 cm. The minimum should be 3/3 and the maximum 15/15. In this procedure use hydrogen peroxide–ethereal only.

The Thermocatalytic Bleach

The thermocatalytic bleach may be most effective. The chamber is packed, as previously described, with cotton pellets, and flooded with hydrogen peroxide–ethereal. A 20 inch source of infra-red light (250 watts, white) is employed. The speculum of the shield is best placed 5 cm. from the foremost portion of the clamp. For each centimeter of distance only 1 minute should be employed. Hence, 3 cm. from clamp

—3 minutes. Always use a timer and have the assistant watch the patient during this procedure. The general arrangement is shown in Figure 2. Use the smallest hole practicable and never lubricate the tooth with silicone, petrolatum, or any similar water repellent agent. After applying the rubber dam, swab the tooth to be bleached with the hypochlorite solution. Repeat this procedure before removing the dam, as it neutralizes the hydrogen peroxide.

If two attempts, a combination of the sustained and thermocatalytic, do not suffice to bleach the tooth, further attempts are futile.

CONCLUDING TREATMENT

1. After bleaching is completed, a sustained 5 per cent aqueous sodium fluoride infiltration should be used. Pack the chamber with cotton and saturate with sodium fluoride solution, and close with a rubber base temporary stopping. The fluoride ion diffuses through the dentin and enamel over a period of a week, thereby rendering these structures less permeable and minimizing the possibility of "fallback" discoloration. The latter phenomenon is essentially exogenous. This technique has been thoroughly tested and adjacent control teeth have shown a marked difference. Where "fallback" is likely, the little additional time required is certainly justified. The procedure is referred to as "permanentizing" the bleach.

2. "Diaphanizing" the bleach refers to the restoration of the degree of translucency characteristic of certain teeth. The author has not found anything superior to the use of a 5 per cent clear acrylic solution in chloroform. The solution is taken up on a wisp of cotton and whirled around the walls of the prepared chamber. A gentle stream of air rapidly volatilizes the chloroform. The chamber is then filled with a blend of slightly lighter colored cement than immediately required. In exceedingly translucent teeth, the chamber should be filled with the "T" shade synthetic porcelain (SSW) if exceedingly bluish, or with No. 20 if unusually light and translucent.

THE FINAL RESTORATION

A cavity is reprepared into the coronal filling material to a depth not ordinarily exceeding 1 mm., or to a depth comparable to the dentino-enamel junction if the surface were intact. Synthetic porcelain restorations and the newer plastic filling materials are frequently used.

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The Management of Accidents Encountered in Endodontic Practice

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Accidents can arise during endodontic treatment no matter how skillful the operator. Care will help to prevent but will not entirely preclude accidents from occurring. The author was watching a television demonstration a few years ago given by a well known endodontist with many years of experience behind him. The rubber dam was in place and a bur was applied to the tooth to prepare a cavity. The operator stopped. He was in apparent difficulty for a moment or two. Then he proceeded again. The audience didn't know what happened behind the scenes, but the reason for the delay was that the head of a brand new carbide bur broke and was stuck tight for a while in the cavity it had prepared. In this instance, the bur head was set free in a minute or so because it broke near the surface of the enamel, but the author had a similar experience which took longer than a minute because the bur head became embedded in the gold of a tooth serving as a bridge abutment. While an accident of this kind is merely time consuming, it can present a serious complication if the instrument is broken in the pulp chamber or within the root canal.

BROKEN BUR IN PULP CHAMBER

The author has reported a case in which a bur fragment remained in the pulp chamber for several months, after which a successful pulpotomy operation was carried out. The patient was a high-strung girl, 10 years of age. While the referring dentist was preparing a cavity, he felt the bur suddenly sink into the pulp chamber, the patient "jumped," and the bur head broke off, remaining within the pulp chamber. The patient would not permit him to proceed further, except to let him seal the cavity with zinc oxide–eugenol cement. Under local anesthesia, 4 months later, the bur fragment was removed and a pulpotomy operation was successfully carried out.

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In another case, a bur was broken in an upper lateral while the dentist was preparing a cavity in order to give the patient relief from pain associated with a subacute abscess. A bur should never be used in a root canal. The bur fragment, removed by the author, was not easy to retrieve. Although a cavity was prepared which was wider than the bur head, the fragment was long and seemed to bind each time an effort was made to dislodge it by inserting a root canal instrument alongside the fragment and teasing it. Finally, after considerable manipulation without success, a few fibers of cotton were loosely wound on a medium-size barbed broach. This was inserted into the

<div align="center">A   B   C   D</div>

Fig. 1. *A*, Bur head and portion of shaft broken off part way up into the root canal. *B*, Attempt at removal of bur head; the instrument by-passes the bur head. *C*, Bur head removed from root canal. *D*, Completed root canal filling.

canal and twisted, then untwisted, until slight tension was felt indicating that the cotton had become wound around the fragment. On gently removing the broach, the bur head came with it.

BROKEN INSTRUMENT IN ROOT CANAL

One of the commonest accidents in endodontic practice is breakage of a root canal instrument. A broken instrument often means an extracted tooth. A few hints, if followed, will help to prevent broken instruments. (1) In using a barbed broach, always select one which can be given a complete turn in the root canal without binding. If the broach binds, it will probably break. (2) Always select a brand new previously unused broach for removing a pulp or pulp fragment. It is both safer and less expensive than running the risk of a broken broach. Save the used broach for removing dressings from the root canal, never for removing pulp tissue. (3) For a given size, files are wider than reamers. Let a reamer of corresponding size precede a file. (4) Both reamers and files are flexible from sizes No. 1 to No. 3, but rather stiff beginning with size No. 4. Where a curved root canal is present, considerable reaming and filing should be done with a No. 3 instrument

in order to overcome the curvature and so permit a No. 4 reamer to be inserted to the apex. To force a No. 4 instrument is to invite breakage, a ledge, or a perforation. (5) Examine all instruments before inserting in the root canal to make sure there is no kink in the broach, that there is no rust on the reamer or file, that the instrument to be used is still sharp, and that the blades are regularly and uniformly placed— not uneven. The last-mentioned is extremely important. If the convolutions on the reamer or file are not uniform, the instrument has already been under tension and the chances for breakage are good. (6) Do not force a root canal instrument if it binds, but remove it at

A   B   C
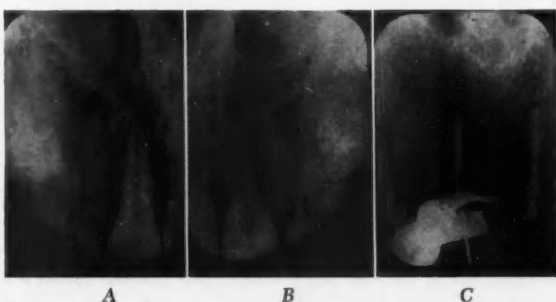
Fig. 2. Examples of broken instruments in root canals. A, Small fragment broken in curved root canal near apex. B, Fragment broken in root canal where canal became very narrow. C, Large fragment broken in canal. Considerable pressure must have been exerted to break such a large size root canal instrument. Such breakage could have been avoided.

once. If the instrument is turned or twisted when it binds, it is likely to break. (7) Never use an engine-driven instrument in a root canal.

A broken root canal instrument may sometimes be removed by inserting another instrument alongside it with the object of loosening the fragment and thus removing it. If a broach is broken, a wisp of cotton on another instrument introduced alongside it may help to entangle the fragment and so remove it. In one case, the author was not successful in removing the fragment by this method but the fragment came out upon irrigating the canal with hydrogen peroxide and chlorinated soda solution. In most cases, no amount of teasing the fragment with an instrument will help to loosen it. A solvent of steel, such as an inorganic acid or a concentrated iodine solution, may be tried but the author has not been very successful in his attempt to dissolve broken instruments. It is generally a waste of time to attempt to dissolve an instrument fragment unless the fragment is very small, say about 1 or 2 mm. only. In a few cases, where the fragment was not broken either at the root apex or through the apical foramen, an

attempt was made to by-pass it by instrumenting alongside it. In other cases, where the fragment was in the apical third of the canal or extended through the apical foramen, root resection was done with recovery of the fragment. The author recalls one case, however, where a rather large fragment of a root canal instrument was broken in an upper anterior tooth. During root resection, it was utterly impossible to pull it out or force it back into the canal even after cutting away at least 4 mm. of the root tip. Under the circumstances, a portion of the instrument was ground away in the canal (from the resected end), an undercut was prepared and the artificial "foramen" so created was sealed with amalgam. In cases where such a procedure is not possible, extraction should be done. In one case where a molar tooth was involved, intentional replantation was carried out after removing the fragment from the resected root end.

LEDGE FORMATION

In the course of instrumentation, a ledge is sometimes inadvertently made owing to the fact that the instrument did not follow the course of the root canal. In such cases, it is often difficult, if not impossible, to retrace one's steps. In one case where the author made a ledge in an upper bicuspid, it was impossible to re-enter the canals to the apex. Since an area of rarefaction was present, root resection by the amalgam technique had to be resorted to, i.e., an artificial foramen was made with a bur and this was filled with amalgam, thereby blocking off the apical 3 mm. of the root canals.

It should be remembered that where a curved canal is seen on a roentgenogram, the root canal instrument should be bent to correspond with the curvature of the canal. The bend should be smooth and gradual, not sharp. Ledges are often made in going from a No. 3 instrument to a No. 4 instrument because the latter is not very flexible.

In some cases where a ledge has been initiated, it might not be possible to retrace one's way up into the root canal with the instrument previously used. For example, a No. 3 reamer had been in the root canal clear to the apex but the No. 3 file did not go as far without risk of creating a ledge. In such a case, it is advisable to return to a smaller size instrument, say a No. 1 or No. 2, and follow with the next sizes serially until the maximal size is reached.

PERFORATION

Perforation of the crown of the tooth while gaining access to the pulp chamber, or of the root while instrumenting in the canal, may

Fig. 3.

A   B

A   B
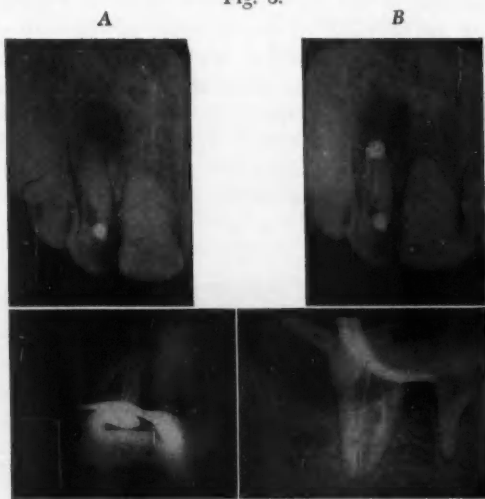
Fig. 4.

Fig. 3. *A,* Root canal instrument broken in canal and gutta percha (?) filling around it (prior to resection). *B,* Following root resection, creating an artificial foramen, and filling the foramen with amalgam.

Fig. 4. *A,* A ledge was created in the mesiobuccal canal of the upper molar which made further penetration of the canal impossible. *B,* The mesiolingual canal of the lower molar is still patent but the mesiobuccal canal is not, as a ledge has been made.

also occur. Where the pulp chamber is almost obliterated, as in an older tooth in which the chamber is greatly reduced in size because of secondary dentin formation, it may be difficult to prevent a perforation. Likewise, a young tooth which has been traumatized may have its chamber almost obliterated by efforts of the pulp to protect itself. An effort to penetrate into the pulp chamber may be fruitless and result in perforation. At times, a tooth may be in such poor alignment, sloping at such an angle, that perforation results unless the bur is properly angulated in relation to the long axis of the tooth. Because of the lingual inclination of the roots of the lower bicuspids and the narrow mesiodistal diameter of the teeth at the cervix, perforation of the crowns of these teeth is not infrequently seen.

Perforation of the root canals is perhaps most often seen in lower molars or of the apical 2 mm. or so of the upper laterals because of the curvature of the root tips of these teeth. Perforation can often be prevented by shaping the root canal instrument to conform to the curvature of the root, i.e., by making a gradual bend at the tip of the instru-

ment. Perforation of the root occurs most often where an engine-driven instrument is used, as the direction of the instrument is not under the control of the operator, nor is the sense of touch present which guides the dentist when operating with hand instruments.

Perforation of the floor of the pulp chamber also occurs at times, especially where the roof and floor approximate each other and the operator has no means of knowing that the roof has already been penetrated because the usual "give" of the bur as it enters the chamber is lacking.
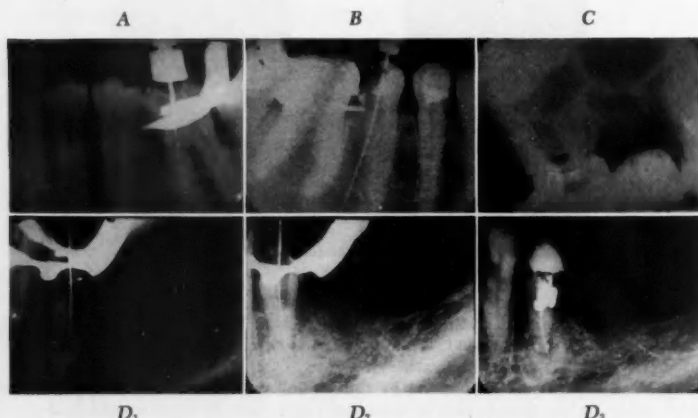
Fig. 5. *A*, Perforation of mesial canal of a lower molar owing to curvature of root. *B*, Perforation of a lower bicuspid with a bur owing to lingual inclination of crown of tooth. *C*, Perforation of floor of pulp chamber of an upper molar. D_1, Perforation of distal wall of a lower bicuspid. D_2, Re-entry into root canal. D_3, Perforation was filled with amalgam while root canal was temporarily obturated with a gutta percha cone, which was later cemented in the canal.

Once the pulp chamber or root canal is perforated, bleeding occurs. This is generally profuse when made by a bur, not only because of the size of the opening but also because of the amount of damage done to the cementum and periodontal membrane with so large an instrument. When perforation is made in the root canal with a narrow instrument, bleeding may not occur. Bleeding from the pulp chamber may be stanched by the application of a cotton pellet saturated with 30 per cent hydrogen peroxide (Superoxol), or with 1 per cent epinephrine solution. Bleeding from the root canal can usually be controlled by alternate irrigation with 3 per cent hydrogen peroxide and 5 per cent chlorinated soda solution. Following the control of bleeding from the root canal, a dressing may be sealed in, and if there are no symptoms and negative cultures have been obtained, the root

canal may be filled with an excess of root canal cement and a tight-fitting gutta percha or silver cone. The latter should be carried into place with pressure in order to force some of the root canal cement into the perforation.

The perforation from the root canal may be large enough, however, to cause much hemorrhage which cannot be adequately controlled. Also, sealing of the root canal causes a painful periodontitis. In such cases, if the perforated area is accessible, root resection is indicated. If it is not accessible, i.e., on the lingual or lateral surface of the root, extraction or intentional replantation may be necessary.

Where perforation of the crown has occurred and the perforated area is accessible, e.g., the floor of the pulp chamber of a molar or bicuspid tooth, the perforation may be filled with amalgam after temporarily plugging the root canals with gutta percha cones. (Note: Do not use silver cones, as the mercury of the amalgam will combine with the silver cone to form a soft amalgam and make removal of the cone from the canal impossible.)

ACCIDENTS DURING IRRIGATION OF THE ROOT CANAL

Root canals are often irrigated with hydrogen peroxide and chlorinated soda solution. Despite care to insert the needle of the syringe loosely in the root canal so as to allow egress of solution, one will occasionally bind the needle in the canal so that the solution is forced through the apical foramen into the periapical tissue. The result is marked irritation of the periodontium. If this accident occurs while the tooth is under the influence of a local anesthetic, e.g., during vital pulp extirpation, and the canal is sealed, considerable pain with edema will result. If it results during the treatment of a pulpless tooth, the patient will react at once to the pain. Treatment should consist in evacuation of the irrigating solution by pulling back on the plunger of an empty irrigating syringe so as to create suction. Absorption of the irrigating solution with absorbent points for at least 5 minutes as it slowly drains back into the canal should follow until the patient is comfortable. The canal may need to be left open for drainage. If pain does not subside, an injection of a local anesthetic may be given not only to control the pain but also to dilute the irrigating solution.

ACCIDENTS ARISING FROM ROOT CANAL MEDICAMENTS

Accidents arising from root canal medicaments may be due to those used for destroying the pulp, sterilizing the canal, or bleaching of a tooth.

While arsenic trioxide should not be used for destroying a vital pulp, it is still used by a few of the older practitioners. Arsenic is a highly irritating, destructive agent when in contact with vital soft tissue. Occasionally, it is sealed in the pulp chamber with less care than its use justifies, and it leaks out of the tooth onto the gingiva. The result is necrosis of the gingiva, and sometimes of the periodontal membrane and bone. Happily, the author hasn't seen a case of this kind in several years.

Formocresol is a highly irritating root canal medicament used for sterilization of the root canal. If used at all, it must be used very sparingly. If the dressing in the root canal is well saturated with the drug, it will diffuse to the periapical tissue and produce a periodontitis. In such cases, removal of the dressing and treatment for marked periodontitis by leaving the canal open for drainage is indicated.

Superoxol is a 30 per cent solution of hydrogen peroxide which is used for bleaching discolored pulpless teeth. Although it will not destroy tissue, it is a highly irritating medicament and care must be exercised in its use. The rubber dam must hermetically envelop the tooth under treatment to prevent leakage. A small hole punched in the rubber dam and a well placed clamp will prevent leakage. During the bleaching operation, the dentist must be careful to direct the syringe, which is used to convey the Superoxol to the tooth, away from the patient so as not to squirt the chemical accidentally on the patient. Also he should be careful not to get any Superoxol on his hands, especially around the nail bed or under the fingernails, as it causes marked irritation and inflammation. If this should occur, thorough washing with water followed by application of a protective agent such as a burn remedy is indicated.

ACCIDENTS DURING FILLING OF THE ROOT CANAL

Accidental overfilling of the root canal may occur. Ordinarily, if the overfilling is slight and the materials used in filling the canal are non-irritating and sterile, no reaction will occur. In some cases, however, overfilling may be excessive enough to cause irritation of the periapical tissue. This is especially true where pulpectomy has been done and the filling material impinges on vital pulp tissue. At times, periapical curettage or root resection may need to be resorted to in order to eliminate the filling material causing the irritation.

Where the sectional method of filling a root canal is used, a fragment of gutta percha may be lost through the apical foramen. As the gutta percha fragment cannot be retrieved through the root canal, periapical curettage will generally be necessary to remove it.

A   B   C
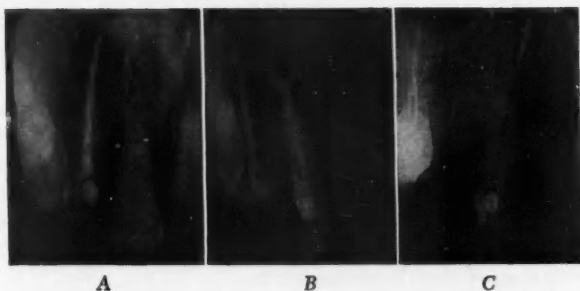
Fig. 6. *A*, Gross overfilling of root canal of an upper lateral. *B*, Bizarre over-filling of root canals of an upper central and lateral. *C*, Loss of section of gutta percha cone while filling a root canal by the sectional method.

EMPHYSEMA DURING ROOT CANAL TREATMENT

Emphysema is the entrance of air into soft tissue. A number of cases of emphysema have resulted from the use of compressed air directed into the root canal for drying it. The resultant swelling is alarming and in some cases the tooth has been extracted immediately in the hope that spontaneous regression would occur. However, such treatment is not necessary as the air is trapped in the soft tissues and will not diffuse out immediately even when a larger outlet is available.

The author has often cautioned students against the use of compressed air for drying a root canal. In one case, a postgraduate student stated that he had used this method for several years without mishap. A few years later he acknowledged his first (and last) case, vowing never to use compressed air again, so frightened was he when the emphysema occurred.

Compressed air may be used for drying the crown of a tooth by directing the stream of air at right angles to the long axis of the tooth, not into the root canal. It may also be used for drying the crown of a tooth after irrigating the canal, preferably after an absorbent point has been placed in the root canal.

ACCIDENTS ARISING FROM ABSORBENT POINTS

One does not ordinarily consider the possibility of accidents arising from the use of absorbent points. Nevertheless, accidents can happen from their use, either from forcing an absorbent point through the apical foramen or from packing it into the root canal. The author has had three cases referred to him where an absorbent point was accidentally lost through the apical foramen. In all cases, the patients were

children. The upper anterior teeth were involved, the root canals and apical foramina were wide, and areas of rarefaction were present. In all three cases an absorbent point with an antiseptic had been sealed in the canal; later, during an attempt to remove the absorbent point, it was apparently pushed farther up into the canal until it went through the apical foramen and lodged in the periapical tissue. In 2 of 3 cases an acute reaction developed that made it necessary to leave the canal open for drainage, while no reaction occurred in the third case. Root resection or periapical currettage was done in all 3 cases in order to remove the absorbent points.

In 2 other cases, the absorbent points were packed into the root canals so tightly that their removal was extremely difficult. Apparently, the absorbent points in each case fitted the root canal rather tightly when dry, later becoming swollen as they absorbed both the medication and periapical exudate. The author removed the absorbent point in one case in a matter of minutes, but in the other it took him more than half an hour of continuous effort with a large number of broaches and other root canal instruments. Efforts to engage the absorbent point so as to remove it in its entirety were fruitless. The absorbent point, embedded in the canal of a lower incisor, was literally picked to bits before it was completely removed.

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